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SPINA BIFIDA AND CRANIUM BIFIDUM*

I. A Survey of Five Hundred and Forty-Six Cases

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DURING the last two decades, 546 infants and children have been seen at the Children's Hospital with spina bifida or cranium bifidum. Associated with these conditions in the minds of both the laity and the medical profession is a rather deep pessimism. It seems worth while to analyze this group of patients, therefore, to see whether there can be established any reasonably definite criteria for prognosis, for operative therapy and for operative technics and whether such an attitude of hopeless pessimism should not be modified by examination of the actual end result in a large series of cases.

INCIDENCE

Race, sex and economic status seem to have little bearing on the incidence of spina bifida. All our patients were members of the white race. Females seem slightly more liable than males, 296 patients being girls and 250 boys. The incidence in the different economic levels seemed proportionate to the total number of patients on each level. The ratio of incidence to the number of new patients seen at the hospital during the last twenty years is 1:4150.

RELATION TO FAMILIAL OR ASSOCIATED ANOMALIES

The familial incidence of spina bifida is shown in Table 1. In 16 (6 per cent) of the 277 cases in which information was available, the patients admitted spina bifida in the family. Approximately an equal number admitted anomalies other than spina bifida.

The frequency with which spina bifida is associated with other congenital anomalies in the same patient is impressive. In 232 patients, or slightly more than half the series, 570 associated anomalies were recorded. Even this large number is probably an underestimate, for two reasons.

TABLE 1. *Incidence of Familial Congenital Anomalies.*

FAMILY HISTORY	NO. OF CASES
Congenital anomaly denied	246
Spina bifida	16
Other anomalies	15
No data	269
Total	546

First, in a number of the records no statement is made concerning the presence or absence of associated lesions; these are chiefly the records of patients who were seen only in the Out Patient Department (often on only one or two occasions) and extensive examination and skeletal x-ray studies were not done and in whom the presence of hernial sacs, genitourinary anomalies or skeletal defects might well have remained undetected. Secondly, many of the internal anomalies can be definitely established only by post-mortem examination, and this was done in relatively few cases.

It is with some hesitation, then, that we give Table 2, fearing that misunderstandings may arise. It must be realized that this is a mere tabulation of all the lesions noted and recorded from such diverse sources as records from the Out Patient Department and autopsy protocols. It is probably incomplete, particularly regarding internal anomalies, and to use it to demonstrate the comparative frequency of the associated anomalies is unwarranted. For example, only 20 cases of Arnold–Chiari malformation are listed. This apparently low incidence of brain-stem anomalies must be largely a factor of the method of the compilation of the data. When the Arnold–

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Chiari malformation is found, in our experience it is usually associated with the presence of a myelocele. The patients with myelocele, however, are usually the poor-risk patients and few come to operation, more being institutionalized or cared for at home, and thus not dying in the hospital

TABLE 2. *Associated Congenital Anomalies.*

ANOMALY		NO. OF CASES
Hydrocephalus		208
Clubfoot		102
Bones		94
Vertebras (including 7 cases of Klippel-Feil syndrome)	49	
Ribs	23	
Skull	16	
Sprengel's deformity	3	
Other bones	3	
Central nervous system		58
Cerebrum	20	
Cerebellum and brain stem (including 20 cases of Arnold-Chiari malformation)	26	
Other portions	12	
Hernia		27
Dislocated hip		23
Pilonidal sinus		13
Genitourinary anomaly		11
Congenital heart disease		4
Other anomalies		30
Total		570

or receiving post-mortem examination. Thus, relatively few patients with myelocele have been autopsied, but of these a high percentage have shown associated cerebellar or brain-stem anomalies. It seems likely that if more autopsy material were available, more such anomalies would have been found.

It will be noted that several of the commoner associated anomalies may have been secondary to the spinal-cord lesions, for example, club feet, dislocated hips and hydrocephalus.

CLASSIFICATION

We have utilized the commonly accepted terminology but have added three subdivisions of our own. We have been constantly impressed by the association of spina bifida and meningeal defects with an overgrowth of fatty tissue. These lipomatous tumors consist of lobules of gritty, firm fat, bound together by fibrous septums, and are usually adherent to adjacent fascial (or dural) planes by firm and often vascular attachments. If a meningeal defect coexists, these tumors may extend intrathecally for some distance, even causing intrinsic compression on the nerves or cord. If such a tumor is present with spina bifida occulta, we have used the term "lipoma." If a meningeal sac is present as well but no nerve elements are involved, we have called the condition a "lipomeningocele." If nerve elements are also involved, we have used the term "lipomyelomeningocele."

All meningeal protrusions, whether or not associated with nerve tissue, whose osseous defect was in the skull (cranium bifidum) we have grouped together as "encephalocele." Simple dermoid cysts arising from the dura and extending through the bone have been excluded.

Often classification of a lesion from the data available in the record is difficult. When surgical or pathological data are available, diagnosis can be accurate, but the clinical distinction between a meningocele and a myelomeningocele is often per-

TABLE 3. *Incidence of Lesions.*

TYPE OF LESION	NO. OF CASES
Spina bifida occulta (13 with lipoma)	65
Meningocele	98
Lipomeningocele	14
Myelomeningocele	279
Lipomyelomeningocele	18
Encephalocele	84
Total	558

plexing. We have therefore adopted the following criteria. If a sac exists clinically and nerve elements cannot be seen, and if there is no evidence of neurologic disability as demonstrated by muscular weakness or paresis, loss of sphincter tone or aberration in cutaneous sensation, the lesion is a meningocele. If nerve elements are visible or neurologic disability exists, the lesion is a myelomeningocele.

With these criteria, the relative frequencies of the different types of lesion are shown in Table 3.

SITE AND AGE

Table 4 records the site in which these lesions were found. Six patients suffered from multiple defects, and 2 of these had three separate lesions.

TABLE 4. *Site of Lesion.*

TYPE ACCORDING TO SITE		NO. OF CASES
Cranial		84
Nasal	5	
Nasopharyngeal	1	
Frontal	6	
Parietal	9	
Occipital	63	
Cervical		23
Thoracic		39
Thoracolumbar		43
Lumbar		205
Lumbosacral		87
Sacral		46
Thoracolumbosacral		10
Undesignated		8
Pelvic		1
Total		546

The age at which the patient was first seen is of some interest (Table 5). As would be ex-

pected, the majority of the patients were seen in the first year of life. In these the local mass or deformity was the presenting complaint. In 42 patients, the chief trouble was orthopedic, whereas

TABLE 5. *Age of Patients when First Seen.*

AGE	NO. OF CASES
Less than 1 week	102
1–3 weeks	180
1–11 months	179
1 year	29
2–4 years	30
5–11 years	26
Total	546

14 complained initially of urinary difficulties. Many of these patients were well along into childhood before increasing neurologic disability led them to seek aid. In 16 patients, spina bifida occulta was found as a fact unrelated to the admitting complaint.

Among the patients with encephalocele there was a great deal of variation in the size, site and amount of neural tissue involved, and these factors are of prognostic significance. If a protrusion consists only of dura containing fluid attached by a pedicle, its size is of slight importance, large ones being as readily removed as smaller ones. If, however, the sac contains a neural mass, and particularly if the ventricular system is demonstrated by pneumoencephalographic studies to extend into this mass, the prognosis is usually poor. Nevertheless, occasionally a sac contains anomalous, abnormal neural tissue that can be excised with impunity. We have found that lesions in the occipital region more frequently contain neural elements and if so offer a poorer prognosis than do lesions in other sites, even though they are small. This is apparently because such lesions are frequently associated with a malformation at the base of the brain, which leads to a block of the flow of cerebrospinal fluid and to hydrocephalus, and the latter proves fatal whether or not the lesion is removed. Associated anomalies of the brain are occasionally found even with apparently hopeful lesions in all sites, so that prognosis must be guarded.

In our operative technic the sac is developed to its base, excised together with its neural contents, and the dura tightly closed. A firm closure of the soft tissues and skin is considered imperative. No attempt is made to repair the bony defect.

The results obtained by operation for encephalocele are, on the whole, encouraging. In our series 59 patients had adequate follow-up studies. Of these, 52 underwent operation, and 17 (33 per cent) of them subsequently died, either in the immediate postoperative period or at some later date. Twenty-one (34 per cent) of the 59 patients are alive, well and *entirely normal*. One of these when last heard from was playing football on his high-school team. When one sees a photograph of this boy as a baby with a sac almost as large as his head protruding from the occiput, this result becomes even more impressive. Other results are equally striking. Table 6 lists the present status of our 84 patients with encephalocele. From these data it seems fair to conclude that about a third of the patients with encephalocele can, with the aid of operative therapy, look forward to an entirely normal existence.

Spina Bifida

Because the complaints and the degree of disability of patients with spina bifida are so varied, classification for analysis of a large group presents

TABLE 6. *Present Status of Patients with Cranium Bifidum.*

STATUS		NO. OF CASES
Alive		37
Operated on	35	
Normal	20	
Mental retardation	6	
Increasing hydrocephalus	4	
Associated anomaly	5	
Unoperated on	2	
Dead		22
Operated on	17	
Unoperated on	5	
Unknown		25
Total		84

many difficulties. It seems as if each patient were almost a law unto himself. It appeared, however, that in the absence of a leak in spinal fluid, with its concomitant danger of infection, there are two major factors that constitute a threat to the patient's life and health: hydrocephalus and injury to, or the lack of, the neural elements of the vertebral canal. The former is recognized by an enlargement of the head, undue separation of sutures and tense fontanelles; the latter by muscular paresis or weakness, lack of sphincter tone and loss of cutaneous sensation. The presence of any one of the latter or a combination of them we have termed "neurologic disability." We have found patients who have had all possible combinations of these two factors, but by utilizing them as a means to classification we can introduce some order into an otherwise chaotic situation. Nor is this division unduly arbitrary, since it parallels, to a certain extent, clinical thought in evaluating these patients as operative risks.

In the operative mortality and postoperative results any series of cases of spina bifida depends on the indications for operation. Does the presence

of increasing neurologic disability — progressive incontinence, for example — constitute an indication for or against operation? Does the presence of hydrocephalus contraindicate operation? What aspects of the local lesion constitute operative indications? On the answer to these questions there seems to be no general agreement. At the Children's Hospital in recent years all cases of spina bifida have been under the care of the Neurosurgical Service and certain concepts of operability have been elaborated.

The presence of a mass constitutes an indication for operation in most cases. Occasionally a small lipomatous tumor is found with occult spina bifida and no neurologic disability, and in such cases operative interference is not necessary. The age of choice for operations in infants is between twelve and eighteen months. This allows time for the development and recognition of disabilities and hydrocephalus, for the local growth of skin adequate to permit closure and for the child to develop in stature and nutrition into a better operative risk. However, certain local considerations may alter the desirability of waiting. If the sac is broken but uninfected, or if it is so thin as to threaten rupture at any moment, and the patient appears to be otherwise relatively normal, immediate operation is demanded to save his life. The presence of infection of the sac or of meningitis contraindicates operation, but local dressings and chemotherapy may suffice to overcome the infection.

Assuming that the mass does not threaten rupture, the patient can be cared for adequately on the following regime. The mass and surrounding skin are thoroughly washed with boric solution and a sheet of perforated Cilkloid applied. A "doughnut" of sheet wadding wrapped in gauze is placed around the lesion for protection. Over the top is placed one or two gauze pads, and the whole is bound to the infant by a flannel or linen binder. The Cilkloid is replaced by the mother as necessary but not too often, since frequent changing nullifies the stimulative effect on epithelial proliferation. The lesion is washed daily with boric solution and clean gauze is reapplied. Every one or two months the patient visits the hospital, where progress is evaluated.

The presence of progressive hydrocephalus constitutes a contraindication to operation. Occasionally hydrocephalus stabilizes, and if this occurs, the contraindication no longer exists.

The presence of neurologic disability does *not* necessarily constitute a contraindication; indeed, particularly in the older age group, if such disability is progressive, we believe that it is a strong argument for operation. In many of these patients, owing to their defect, the spinal cord becomes anchored at this site. As the child grows tension is placed on the cord, since it increases in length less rapidly than the vertebral canal. There may also be local compression by lipoid tumor growth. Both these elements contribute to the development of the signs of nerve dysfunction. This usually occurs between the ages of six and ten. In such cases, the wisest procedure is exploratory laminectomy with a view to alleviating, if possible, the local situation. This attempt is occasionally successful.

The decision to operate having been made, certain technical phases of the operative procedure are of paramount importance. The skin preparation must be wide and thorough. We use a colored chemical disinfectant, such as diluted tincture of iodine or Tincture Merthiolate, as the final step in the preparation, thus making the cleansed field visible and assuring a wide enough area. The incision, which is usually elliptical, is made with the long axis transverse. Where the lesion is low, the incision must be placed high enough to avoid the intergluteal fold. We always wall off the lower limit of the field from the region of the anus by a sheet of sterile gutta percha before the drapes are placed, and postoperatively by a sheet of rubberized silk attached by adhesive strapping. The sac is traced to its emergence from the bony canal, as much as possible is excised, and a tight closure of the dura is made. For this we use interrupted sutures of fine silk, and make the closure as near watertight as possible. Meticulous care must be taken not to cut or injure nerve elements that may be adherent to or contained within the sac. If need be, these are carefully dissected free and returned to their position within the canal. No attempt whatsoever is made to repair the bony defect. If the cut edges of the fascia of the erector spinae muscle come together easily, they are approximated, but the repair of this layer is of no great importance, and under no circumstances should such great effort be made as to cause compression on the underlying dural closure. A firm repair of the subcutaneous tissues and skin is of great importance. The skin must be made to approximate without undue tension by means of wide undermining of flaps and even relaxing incisions at a distance, if such are necessary. Silver foil is placed over the wound, and a compression bandage applied. For this we have found several layers of gauze held in place by Elastoplast bandage quite satisfactory. For several days postoperatively the patient is held prone on a Bradford frame with the head end about 10 cm. lower than the foot. This position probably minimizes the pressure of cerebrospinal fluid on the dural suture line during the healing period, and thus reduces the possibility of the

development of a leak. Unless there is some indication for doing so, the wound is not disturbed for six or seven days, at which time the sutures can be removed.

Table 7 gives a somewhat detailed listing of the present status of our 462 patients with spina bifida.

TABLE 7. *Present Status of Patients with Spina Bifida.*

			No. of Cases
Alive			234
Unoperated on			74
Normal		11	
With spina bifida occulta	8		
With associated small lipoid tumors	3		
Abnormal		63	
Awaiting operation; no neurologic disability	3		
Awaiting operation; neurologic disability	17		
Operation not advised	38		
Neurologic status normal (associated anomaly)	5		
Operated on (Children's Hospital)			148
No hydrocephalus and no neurologic disability preoperatively		84	
Entirely normal	45		
Developed hydrocephalus (now stabilizing)	5		
Developed neurologic disability (Severely incapacitated, 8; mildly incapacitated, 15; disability immediately postoperatively, 10; disability months or years later, 13)	23		
Developed both hydrocephalus and neurologic disability	4		
Normal as regards spina bifida; associated anomaly	7		
No hydrocephalus but neurologic disability preoperatively		48	
Improved by operation	9		
Made worse by operation	4		
Neurologic disability persists (severe, 25; mild, 6)	31		
Developed hydrocephalus	4		
Hydrocephalus but no neurologic disability preoperatively		7	
Arrested (normal, 1; mild neurologic disability, 5)	6		
Progressing	1		
Hydrocephalus and neurologic disability preoperatively		9	
Arrested (severe, 7; mild neurologic disability, 2)	9		
Progressing	0		
Operated on elsewhere (severe, 10; mild neurologic disability, 2)			12
Dead			167
Unoperated on			127
Operated on			40
Operative deaths (meningitis, 12)	22		
Subsequent deaths (hydrocephalus, 16)	18		
Status unknown			61
Total			462

Any further simplification, however, would deprive it of analytic value.

A portion of this material is presented in condensed form in Table 8, which gives only the present status of the living patients, grading them as normal, slightly handicapped or severely handicapped.

The data presented in Tables 7 and 8 allow some interesting conclusions. There are 401 patients whose status is known and whose follow-up studies are considered adequate. Of these, 234 (58 per cent) are alive. The remainder have died of a variety of causes. Operation was performed on 188, and 20 more are awaiting operation. Thus, 208 (52 per cent) were or are thought to be suitable for operation. The immediate operative mortality in our hands was 12 per cent.

Of the patients who survived, 60 are considered normal and 61 are suffering from neurologic

TABLE 8. *Present Status of Surviving Patients.*

Patients	Normal	Mild Neurologic Disability	Severe Neurologic Disability
Operated on (Children's Hospital)	46	42	60
Others	14	19	53
Totals	60	61	113

mild disability. The patients in both these groups may be considered as capable of leading a normal human existence. Thus, about 30 per cent of the patients with spina bifida may look forward to a life unhampered by any significant incapacity resulting from their anomaly.

Congenital lack of neural elements or destruction by long-standing compression results in neurodisability of an irreversible nature. One of these situations prevails, unfortunately, in the majority of cases where disability is present. However, occasionally local compression or traction on the cord is the cause of the progression of symptoms. If these are recognized, laminectomy offers promise of relief if the local situation permits operation. In our series there were 10 patients in whom relief or improvement in neurologic disability occurred following such an exploration.

In the light of the data presented by this review, we believe that an outlook of extreme pessimism in the presence of spina bifida is unwarranted. Each patient must be evaluated as an individual problem.

Summary

A series of 462 cases of spina bifida and 84 of cranium bifidum (encephalocele) is presented, with data on the incidence, sex, race, type, site, associated anomalies and clinical manifestations.

Therapeutic indications and end results are tabulated and discussed.

Thirty per cent of patients with spina bifida and 34 per cent with encephalocele may expect a relatively normal life. An unduly pessimistic prognosis is therefore unwarranted until individual evaluation has been carefully pursued.

SPASMODIC RECTAL PAIN

A Review of the Literature and Report of a Case

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SPASMODIC pain in the rectum, which is localized a short distance above the sphincter ani, has received scant attention in the literature. It appears without warning, is slightly gnawing at first, but increases steadily in intensity to a point that may cause loss of consciousness, then slowly disappears, leaving no ill effects except a sense of fatigue. The average attack lasts for about fifteen minutes. The patient has a sensation of uneasiness at the anus, with a tightening sensation at the anal canal. A continuous dull ache is felt in the rectum, as though it were ballooning out. This neuralgic type of pain is not related to the intake of food, the type of food ingested or bowel function. It is about twice as common in men as in women.

The commanding character of the pain and the unknown etiology entitle the condition, often called "proctalgia fugax," to be considered a definite entity. As early as 1917, MacLennan,[1] of Glasgow, gave an excellent description of the condition. Later Thaysen,[2] of Copenhagen, and Stolte[3] wrote articles that added much information and stimulated many physicians to send letters concerning cases that had come to their attention. Many of these letters were published in the *Lancet* during 1935.[4] These represent many opinions regarding the etiology of the disease, but not one contains a description of the sigmoidoscopic examination during the attack. The following case report is therefore of interest because I was able to perform such an examination.

CASE REPORT

T. H. S., a 60-year-old man, in good general physical condition, had suffered repeated attacks of rectal pain, occurring at intervals during the previous 4 years. The pain, gnawing in character, awakened him from a sound sleep. It began gradually, and the rectum seemed to be slowly ballooning out. The pain increased in severity and seemed to involve an area in the rectum "about 5 inches long." Slight spasm was noted in the area of most intense pain. Regardless of change of position, the patient continued to suffer acute distress. Efforts at micturition and defecation were unavailing, although the patient stated that he thought he would obtain relief if he could pass urine or gas.

After 15 minutes or so, the pain gradually subsided. The patient felt no ill effects except a feeling of lassitude. He was able to go to sleep readily after each attack, and had no subsequent awakening during the remainder of the night. Attacks came at intervals of about three months, but never in the daytime.

During an attack on July 26, 1942, a rectal examination was made, and proved enlightening. Digital examination revealed slight rigidity of the muscles of the rectum, and the mucosa of the anal canal felt hot, as though inflammation were present. The pain persisted. The prostate gland was enlarged. Insertion of the anoscope revealed the presence of a few small hemorrhoids. The mucosa was bright red.

Through the sigmoidoscope the rectal mucosa appeared redder than normal, and resembled that seen following prolonged or rough massage of the prostate gland. It seemed swollen, and the vessels were more prominent than normal. The site of the pain was at the central portion of the levator ani muscle, and slight pressure exerted by the tip of the instrument at this point caused mild spasm. No ulcerations were noted. The middle valve of Houston was more rounded than normal and the superficial vessels more pronounced. As the instrument reached the rectosigmoidal angle, some difficulty was encountered in locating the lumen of the bowel. At this point, with the passage of a considerable amount of gas, the patient noted relief of the pain. As the sigmoid was approached, normal mucosa came into vision. Prostatic massage was done, and repeated three days later.

Sigmoidoscopic examination a week after this attack showed the mucosa of the anal canal to be still bright red. The rectal mucosa was pink, much paler than when seen during the painful episode. The veins were not engorged. In fact, the picture was that of a normal rectum.

In all, 12 cases of spasmodic rectal pain have come under my observation — 10 in men and 2 in women. All the men had prostatic hypertrophy and both women had pelvic inflammation. Many hypotheses concerning the etiology of the condition have appeared in the literature, but any connection between rectal spasm and prostatic hypertrophy in the male or pelvic disease in the female has for the most part been disregarded. Beckman[4] stated in passing that his patient had had prostatic massage before his attack, but ascribed the condition to excessive smoking. The seizures simulated those occurring in certain types of "pseudoangina," which clear up when the patients forego the use of tobacco.

All authors agree that proctalgia fugax is a rectal rather than an anal phenomenon. Attacks occur at night, as a rule, and during periods of fatigue and stress. No rectal disease is found, although in some cases hemorrhoids are present. The pain is similar to that of tension, contraction or even incision, accompanied by a sensation of pressure. The attacks are not followed or preceded by a desire to

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defecate. As a rule, if they are frequent they are less severe. In 2 of 9 cases reported by Thaysen,[2] there was a family history of the condition, one or both parents suffering similar attacks.

Stolte[3] believes that the pain is of nervous origin and is due to spasm. It does not occur in the sphincter ani, and anal spasm is always secondary. All patients are sure that the pain is above the anus. According to Smith,[4] when the examining fingertip touched the fibers of the levator ani muscle, the patient said, "That's the spot where I get the pain."

Thaysen[2] and others consider it a spasm of the rectal musculature. MacLennan[1] and Abrahams[4] believe that the lesion is the result of a neurosis, although it affects many persons of a non-neurotic temperament. Smith[4] states that pain is due to a cramp of the levator ani muscle and gives the following reasons: the pain is analogous in character to muscle cramp elsewhere in the body; it has a tendency to spread and to involve the entire muscle; it occurs during periods of fatigue; it is prone to arise in a muscle that has recently been excited; it appears when the patient is getting warm in bed, as is the case with cramps in other muscles; and victims have obtained relief by stretching while assuming a squatting position.

Ryle[4] associates the attacks with mental or physical fatigue. Mummery (quoted by Ryle) regards the disease as a tic. The pain has been associated with sexual excitement, masturbation, migraine and epilepsy (Carlill[4]). A woman patient stated that attacks always occurred just before or just after menstruation (Tecon[4]), but no pelvic disease could be discovered. Yeomans[5] reported 2 cases, both in women. No pelvic disease could be found in one; the other patient had been operated on for anal fissure, with a deep incision through the levator ani muscle in the posterior commissure. Thus a nerve filament might have been caught in the extensive scar. However, no relief followed when the scar was dissected out.

A review of the innervation of the rectum and anus in the light of the symptomatology indicates that the site of the pain is in the rectum, as all the patients insist. The tissues above the pectinate line are supplied entirely by the sympathetic system, whereas below, in the anal region, the tissues are supplied by spinal nerves and also by some fibers from the sympathetic nerves. Above the line (in the rectum), the ordinary sensations such as touch, pain and heat or cold are not found to any degree, and usually are not present at all; but there are sensations of pressure and muscle sense.

Inspection of the anal area frequently affords considerable information concerning whether the irritation is in the anal segment, innervated by the spinal nerves, or in the rectum, innervated by the sympathetic nerves. With infection, irritation or trauma of the portion below the anorectal line, spasm of the sphincter muscles and contraction of the anal aperture are the rule. A rectal lesion — that is, one above the line — is frequently associated, not with spasm of the muscles, but with a patulous condition and relaxation of the anal ring (Martin[6]).

Inflation of the rectum with air has been advocated by Ryle,[4] Marshall[4] and others. Locally, applications of heat, as by a sitz bath or enema, and the insertion of various kinds of suppositories have been employed with good effect. Mummery[4] uses alcohol injection, since he believes the condition to be a tic of the sacral plexus. Yeomans[5] injects 30 cc. of a 1 per cent solution of novocain into the sacral canal, followed by 30 cc. of 25 per cent alcohol if the relief is only temporary. Moltke (quoted by Thaysen[2]) suggests amyl nitrite, because the nitrites have been employed successfully for other spastic affections of the bowels, and also because, if efficacious, it would prove that the condition is primarily due to spasm. Tecon employs short-wave diathermy. Beckman[7] mentions the possibility of an allergic factor and points out that in 2 cases Rowe's elimination diets were of help. Often, however, the attack ceases spontaneously before the patient has had time to try any specific remedy. No sequelae have been noted, and the condition has a tendency to improve with advancing age. For this reason Hanssen (quoted by Thaysen[2]) classified proctalgia fugax as a minor complaint, but admitted that attacks might be severe enough for it to be considered a distinct entity.

Differential Diagnosis

The attacks of proctalgia fugax are different from the crises of tabes dorsalis, and indeed no patient with this complaint has been tabetic. The former attacks are fleeting, occurring at widely spaced intervals. Those of tabes are accompanied by watery and frequent stools, hemorrhage from the anus and tenesmus. The pain radiates, whereas in proctalgia fugax, as Ryle[4] and Stolte[3] have demonstrated, it is definitely localized in the levator ani muscle.

Proctalgia fugax also differs from the nervous type of rectal pain called enteralgia, or neuralgia of the mesenteric plexus. In the latter condition, attacks may last for several days, the pain radiates, and the attack is often preceded or followed

by abdominal pain. It may be part of a syndrome due to lead colic, colitis or even duodenal ulcer.

The pain differs also from that of coccygodynia. In the latter, patients are most uncomfortable in the sitting posture, and there is a dull ache at the base of the spine. Manipulation and massage improve the condition. With proctalgia fugax, the patient is loath or unable to move because of the excruciating spasm. Also proctalgia fugax is commoner in men, but coccygodynia in women.[8]

A cardinal reason for establishing the correct diagnosis in these cases is the need of reassuring these patients that they do not have cancer of the rectum. Sigmoidoscopic examination will dissipate this fear and give them confidence. Therefore, even though the symptom-complex of proctalgia fugax is not treated by such authorities as Nothnagel, von Bergmann, French, Babcock, Naegeli, Buie and others (as mentioned by Thaysen[2]), the condition merits more attention, and is commoner than the reports in the literature lead one to believe.

Summary

A case of spasmodic rectal pain is reported, and the etiology, diagnosis and treatment of this condition are discussed.

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CLINICAL NOTE

ACUTE HEMOLYTIC ANEMIA FOLLOWING SULFADIAZINE*

Report of a Case

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ACUTE hemolytic anemia is an important toxic effect following treatment with the sulfonamides. The incidence following sulfanilamide is 1 to 2 per cent and following sulfapyridine 2 to 3 per cent.[1] It is apparently quite rare following sulfathiazole. Spink[2] reported that he had not encountered acute anemia in over 400 patients treated with sulfathiazole. So far as I am aware, acute hemolytic anemia following sulfadiazine has not yet been reported.

Case Report

L. F., a 65-year-old woman, entered the Evans Memorial on September 7, 1942, complaining of dyspnea of 4 days' duration. She had been subject to attacks of "bronchitis" with cough and fever each winter for many years but had had no dyspnea, chest pain or edema. She had never taken any of the sulfonamides. The present illness began rather abruptly with increasing dyspnea and asthmatic breathing accompanied by rapid irregular heart action.

Physical examination revealed an obese woman in moderate respiratory distress. The heart was enlarged to the left, the rhythm was grossly irregular at a rate of 130 a minute, and there was a moderately loud apical systolic murmur. The blood pressure was 230/140. There were moist rales at both lung bases but no venous distention, liver engorgement or edema.

The patient improved satisfactorily on the usual regime of rest, sedation with barbiturate, digitalis and intravenous Mercupurin. On the 11th hospital day, after a completely afebrile course, her temperature rose, she complained of slight pain in the right lower chest, and the respirations rose to 32. There was no cough or hemoptysis. Examination of the chest revealed a moderate number of coarse crepitant rales at the right lung base. A roentgenogram showed patches of increased density at the right lung base, which were interpreted as either a pneumonic process or multiple infarcts. The white-cell count rose to 17,000. Because of the history of recurrent bronchitis and uncertainty concerning the etiology of the lung disease, she was started on sulfadiazine by mouth. The dosage of the drug and the patient's clinical course are shown in Figure 1.

Two days after the institution of sulfadiazine therapy the erythrocyte count had fallen from 4,200,000 to 2,200,000, with a corresponding fall of the hemoglobin from 12.5 to 8.1 gm. The patient became slightly icteric, but this might well have been assigned to the pulmonary infarction, which was subsequently clearly indicated by the appearance of hemoptysis and signs of phlebothrombosis of the right leg. There was no striking pallor to give warning of the precipitous drop in erythrocyte count or hemoglobin. The liver and spleen were not palpable, and there was no nausea, vomiting or skin eruption. The white-cell count showed neither a tendency toward leukopenia nor one toward the hyperleukocytosis seen in some cases of acute hemolytic anemia following the administration of sulfonamides. Despite increasing amounts of fluid the urinary output fell and there was a slight rise in the blood nonprotein nitrogen, but there was no hematuria or crystallinuria. The drug was discontinued, fluids were forced, and the patient was given

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a transfusion of 500 cc. of whole blood. Meanwhile, slight tenderness had been noted in the right calf. The femoral vein was opened and a large loosely adherent clot removed, following which the vein was ligated. The erythrocyte count and hemoglobin value quickly returned to the previous levels, and the subsequent course was satisfactory.

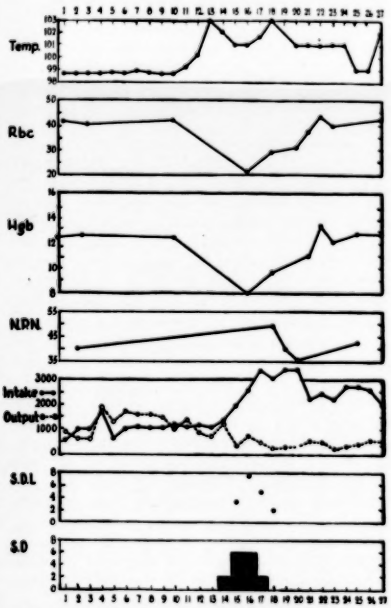

FIGURE 1. *Course of Acute Anemia Developing during the Administration of Sulfadiazine.*

The temperature is expressed in °F.; the red-cell count, in number $\times 10^5$; the hemoglobin in gm. per 100 cc.; the nonprotein nitrogen, in mg. per 100 cc.; the intake and output in cc.; the sulfadiazine blood level, in mg. per 100 cc.; and the sulfadiazine dosage, in gm.

Although sulfadiazine is undoubtedly less toxic than the other commonly employed sulfonamides, vigilance during its administration must not be relaxed. The present tendency to widespread use of sulfadiazine without adequate laboratory control, in the home, for example, is certain to result in tragic consequences sooner or later. In the present case there were no alarming clinical manifestations to warn of the serious, acute anemia. Certain of the toxic reactions may develop very rapidly in the first few days of treatment, and it is probable that as more of the population becomes exposed to the sulfonamides, accelerated reactions due to the development of hypersensitivity to the drug will become commoner. Sulfadiazine had been used in the treatment of over 250 patients in this hospital before this case was encountered. At best, this affords a rough estimate of the incidence of acute hemolytic anemia, but it suggests that it may be no less and possibly somewhat more frequent following sulfadiazine than following sulfathiazole therapy.

The oliguria and elevated blood nonprotein nitrogen in the present case may also have been a direct toxic manifestation of sulfadiazine, but in the absence of hematuria or crystallinuria it seems more likely that the renal complications were a manifestation of hemoglobinuria, although there is no proof that the latter occurred. It is probable that in rare cases the sulfonamides may produce oliguria without signs of renal irritation and in the absence of significant crystallinuria.

SUMMARY

A case of acute anemia following the administration of sulfadiazine is reported.

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MEDICAL PROGRESS

HERPES ZOSTER

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THE accumulated evidence supports the opinion that herpes zoster is essentially an acute posterior poliomyelitis[1] due to the virus of chicken pox and unrelated to the virus of herpes simplex. Although anatomically the root ganglions are placed outside the spinal cord, they consist of cells that have migrated from the posterior horn, and in this way their constant involvement may be accounted for. The inflammatory changes found in the anterior horn by Lhermitte[2] and others are presumably due to the spread of the virus from the posterior horn, and explain the occasional occurrence of muscular paralyses[1, 3] in a disease that primarily involves the sensory system.

PATHOLOGIC PHYSIOLOGY

The pathologic physiology of herpes zoster involves a consideration of all agents that may produce any type of injury to the dorsal-root ganglions. These include pressure of tumors, traumatic injuries, exudates of systemic diseases (syphilis), poisoning by heavy metals (arsenic), leukemia and other blood dyscrasias, Hodgkin's disease and vertebral diseases. It has been suggested that these conditions are only predisposing causes, for the zoster follows its normal course, heals and does not recur despite the continued presence of these factors.[4]

The mechanism of production of the distinctive skin lesions of herpes zoster is of considerable clinical interest. The pathologic physiology involved has been well understood for many years but deserves a brief review. Wright[5] has recently given an admirable summary of this subject. Stimulation of the peripheral end of the cut posterior nerve root produces vasodilatation of the corresponding segments of the skin and muscles. According to Lewis,[6] the vasodilator fibers in the sensory nerves act by liberating a substance ("H-substance") resembling histamine in its properties. It is suggested (arguing from the analogy of the action of histamine) that the H substance dilates the capillaries by direct action on their walls, but that the arteriolar dilatation is due to an axon reflex. The H substance stimulates sensory nerve endings in the skin and impulses pass back along afferent nerve fibers, but the impulses make none of the usual circuits through the central nervous system, and after traveling only a short distance reach branching nerve-fiber junctions and turn peripherally again along branches that reach and dilate neighboring blood vessels. The sensory fibers arising from pacinian and other nerve endings in the skin have been shown to extend numerous collaterals to the adventitia of the neighboring arterioles, and these fibers are believed to be responsible for the dilator effects just described.

To continue one's reasoning along the above lines, in herpes zoster, injurious influences, such as inflammation, hemorrhage, pressure and poisons, act on the posterior-root ganglions or their cranial homologues. These processes lead to stimulation of the ganglions and have an effect equivalent to stimulation of the peripheral ends of the cut posterior nerve roots. As a consequence, dilatation of blood vessels occurs in patches along the distribution of the involved sensory nerves. Antidromic impulses traveling along these nerves release the H substance, which dilates the capillaries and sets up an axon reflex that in turn dilates the arterioles. The H substance not only dilates the minute blood vessels but also increases their permeability. This increase causes the exudation of serum and formation of vesicles within areas of erythema (vasodilatation) along the distribution of the posterior nerve roots of their cutaneous sensory branches.

Head and Campbell[7] as well as Lewis and Marvin[8] believe that the eruption of zoster is due to antidromic impulses passing down sensory nerves to the skin, as just described. Stern,[9] on the other hand, has proposed the theory that the eruption of zoster is due to the passage of causative organisms down sensory nerves to the skin. In support of this interpretation may be quoted the work of Kundratitz,[10] Bruusgaard,[11] Paschen[12] and Amies,[13] who have produced evidence that a virus is present in the vesicles of zoster.

The skin eruption of herpes zoster may be explained by the above physiologic mechanisms. The essential unit is a cluster of vesicles situated on a basic plaque of erythema. Several such clusters

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are usually present and may involve any part of the body. The vesicles themselves are ordinarily tense and thick walled.

The pain of herpes zoster is easily accounted for on the basis of inflammation or degeneration of sensory-nerve fibers entering the involved dorsal-root ganglions. This pain may be severe or may be entirely absent. It may precede and accompany the skin manifestation and, especially in elderly patients, it may occur as a severe and intractable neuralgia persisting for months or years after disappearance of the skin lesions (post-herpetic neuralgia).

Herpes zoster is especially frequent in persons who are overworked or ill. The appearance of the eruption is usually preceded by malaise, neuralgic pain and hyperesthesia of the affected area. The disease runs an acute course lasting from ten to thirty days. One attack almost always confers immunity. Second attacks may occur but are very rare. Many kinds of irritation and damage can precipitate an attack of zoster, "possibly through activation or stimulation of the dormant virus or through reduction of the delicately adjusted factors conferring host immunity or local resistance."[14] The commonest "trigger factors" include the following: drugs, such as arsenic, lead, bismuth, mercury, iodides, gold, morphine, carbon dioxide and carbon monoxide; blood dyscrasia, especially leukemia; trauma; infectious diseases, such as influenza, encephalitis, erysipelas, tuberculosis and syphilis, particularly early cardiovascular syphilis, paresis and tabes; vaccination; and malignant tumors and Hodgkin's disease, which act either by pressure on the ganglions or by toxic effect.

Diagnosis

The typical case of herpes zoster presents no diagnostic difficulties. If, however, the vesicles are confined to some obscure locality such as the ear canal or bladder, and if they remain undetected, it may not be possible to diagnose the disease on the basis of pain and other symptoms. Some observers believe that zoster may occur without skin manifestations. In such cases, the diagnosis may be difficult to distinguish from other types of neuralgia, although a lumbar puncture disclosing the characteristic zoster lymphocytosis of the spinal fluid may be of considerable help. In the early stages of zoster one may encounter cases where there is erythema but no pain or vesicles. Here the diagnosis is not always easy, but the patchy character and unilateral distribution of the erythema should be helpful. The characteristic vesicular plaques of herpes zoster usually appear suddenly and acutely — either one by one or all together. The eruption may take three or four days to become complete, but sometimes new plaques appear as long as two weeks after the onset. Lymph-node enlargement is frequent.

In the differential diagnosis of herpes zoster it is well to remember that the pain may precede the appearance of cutaneous changes and may lead to errors of diagnosis. It may simulate the pain of pleurisy, gall-bladder colic, kidney-stone colic, appendicitis, myositis or neuritis.[15]

The specialist must recognize not only the usual type of herpes zoster but its less common variations such as the gangrenous, hemorrhagic, bullous, bilateral and generalized types.[15] In the gangrenous type, necrotic and destructive lesions occur in what is usually a severe form of the disease. This may happen in any region but is commonest in the ophthalmic area, the cornea frequently being severely involved. Bilateral zoster may be symmetrical or asymmetrical; it is extremely rare and, contrary to lay belief, is not fatal. Excellent illustrations of bilateral herpes zoster are given by Sutton and Sutton.[16] Bilateral herpes zoster involving both trigeminal ganglions has been described by Campbell.[17] Extensive herpes zoster in patients suffering from rheumatoid purpura has been reported by Audibert and Paganelli.[18]

Thirty-two cases of generalized zoster in association with leukemia have appeared in the literature. The first such case was reported by Fischl[19] in 1913. The most recent of these cases have been reported by Wile and Holman,[20] who analyzed all the cases previously described and tabulated their salient features. They found that in 93 per cent the leukemia was lymphatic in type, and that in 71 per cent the patients were men averaging sixty years of age.

Generalized zoster may also occur without association with leukemia. In this interesting form of the disease one finds scattered discrete papular and vesicular lesions, which are usually accompanied by headache, fever and malaise. There are often mucous-membrane lesions and the entire picture is quite similar to that of chicken pox. Only epidemiologic considerations and the presence of a concomitant typical herpes zoster along the course of a nerve permit differentiation from ordinary chicken pox.[15, 21]

Besides the bilateral and generalized forms of herpes zoster, another rare variation is the unilateral type with multiple lesions. Because of its rarity, Walls[22] has reported an example. Most of the reported cases with multiple lesions have occurred in association with severe constitutional illnesses, such as leukemia, Hodgkin's disease and tuberculosis. Presumably in these cases the virus

of zoster may find a vulnerable ground to work on. Walls's patient, on the contrary, was a boy of nineteen in excellent health. He exhibited a typical well-marked, grouped, vesicular rash sweeping around the left side of the chest in the segmental distribution of the sixth dorsal segment. In addition a narrow strip of grouped vesicles extended from below the tip of the acromium down the outer front of the left arm, corresponding to the skin distribution of the fifth and sixth dorsal segments. Furthermore, a similar rash on the left thigh corresponded anatomically with the skin distribution of the third lumbar segment. Finally, there was a group of vesicles on the outer half of the left popliteal fossa traveling down the outer aspect and front of the leg, corresponding to the skin distribution of the fifth lumbar segment. This patient therefore presented four distinct areas of skin involvement, a distribution equally as bizarre as the spinal-cord involvement must have been. The fourth lumbar segment was completely missed but the ganglions both above and below were involved. The patient's general condition was excellent, there being no pyrexia or changes in the nervous system; in fact, he was not sufficiently ill to require hospitalization.

Herpes zoster may occur in childhood. Koch[23] reported a case in a boy of five and a half and Batignani[24] one in a newborn infant whose mother had the disease at parturition. Bruce[25] reports that ocular complications occur in about 50 per cent of all cases in childhood, and he has made an interesting review of herpes zoster of the eye in childhood. He describes in detail the morphology of the eruption as it affects the cutaneous surface of the eyelids, the bulbar and palpebral conjunctiva and the cornea itself. The cornea loses its sensitivity early in the course of ophthalmic zoster. Minute subepithelial dots appear, often coalescing to form large areas of infiltration. Subsequent scarring may lead to serious impairment of vision. The iris is always inflamed if keratitis is present, and this iritis may actually be due to localized herpetic eruptions on the surface of its stroma. In such cases the inflammation is apt to be severe, glaucoma may supervene, and the eye may be lost.

In childhood zoster of the eye, the ocular muscles are attacked singly or in groups in about 7 per cent of all cases, according to Bruce.[25] Complete ophthalmoplegia may be accompanied by exophthalmos. This has been ascribed by some writers to atony of the muscles, and by others to sympathetic stimulation.[26] Ptosis may be either apparent, produced by the weight of the swollen lid, or real, as a result of third-nerve paralysis. Accommodation is paralyzed in some cases. As a rule, the prognosis is good and muscular balance is restored in about six weeks. The optic nerve is rarely involved, but if it is, a severe neuritis followed by atrophy may result.[27]

Herpes zoster following lumbar puncture has been described by Carter.[28] In a case of syphilis of the central nervous system, a lumbar puncture was performed. The needle was passed with difficulty and with much pain in spite of local anesthesia. Eight days following lumbar puncture, the patient developed typical herpes zoster in the left lumbar area and buttocks, later spreading to the inguinal region and anterior upper third of the thigh. There was severe lumbar pain. Recovery took place in one week following the intravenous administration of sodium iodide and x-ray irradiation from the lumbar puncture site to the twelfth dorsal vertebra. In view of the absent constitutional symptoms and the rapid recovery without antisyphilitic treatment, a neurotropic virus and syphilis were not considered possible etiologic factors and the lesion was assumed to have been due to trauma by the lumbar puncture needle. Judging from the localization of the skin eruption, traumatic irritation must have been transmitted to the first and second lumbar dorsal-root ganglions, resulting in an eruption along the dermatomic distribution of the corresponding nerves.

It has been recognized for many years that attacks of herpes zoster may be precipitated by the administration of arsenic. Four such cases have been reported recently by Knowles and Bolton.[29] In one case the zoster developed following the sixth of a series of neoarsphenamine injections. The eruption was distributed over the left arm and disappeared one week following cessation of arsphenamine injections. In the second case zoster appeared on the right upper arm sixty-seven days after the institution of therapy by arsenic trioxide and protiodide of mercury, which were given by mouth in the management of a case of lichen planus. The third patient developed herpes zoster in the left occipital region subsequent to the use of Fowler's solution in the treatment of psoriasis. In the final case, zoster appeared on the right buttock following the twenty-fourth injection of bismuth and Mapharsen in the treatment of syphilis. Knowles and Bolton believe that arsenical herpes zoster is due not to overdosage but to idiosyncrasy.

Complications

As already pointed out, the inflammatory process of herpes zoster may spread from the posterior-horn cells to the anterior-horn cells, with production of motor lesions. Carter and Dunlop[1] discuss in admirable detail the subject of paresis fol-

lowing herpes zoster and report 2 cases. A synopsis of their paper is as follows:

Motor lesions occur infrequently in herpes zoster, although both upper and lower motor-neurone palsies have been described. Upper motor-neurone lesions were described by Besta and Vercelli (quoted by Rocchi[30]), Bruce[31] and Worster-Drought and McMenemey.[32] In each of these cases there seemed to be no other explanation for the pyramidal lesions. Among the lower motor-neurone palsies the one most often encountered is facial paralysis. Occasional deafness complicating geniculate zoster occurs. The next most frequent palsy appears to be oculomotor paresis complicating trigeminal zoster. Cases of this type have been described by Essen-Möller,[33] Federici,[34] Carmody[26] and others. Brachial and abdominal palsies are much less common. Lower motor-neurone paralysis complicating lumbar or sacral zoster is rare.

Carter and Dunlop have described 2 cases of lumbar zoster, both complicated by lower motor-neurone paresis involving the affected segments. These two cases may be summarized as follows:

Case 1. Two months following an attack of shingles in the right leg, a 73-year-old widow began to feel shaky and complained of neuralgia and weakness of the right leg. Examination revealed recent herpes zoster scars along the distribution of the third and fourth lumbar roots. The right quadriceps was wasted and its power as well as that of the thigh adductors was diminished. All the tendon reflexes were normal except for the right knee jerk, which was completely absent. The spinal fluid was normal except for the cell count, which showed 21 cells (20 lymphocytes and 1 polymorphonuclear cell) per cubic millimeter. The patient was treated by a course of faradism and by exercises of the affected muscles. After 3½ months the spinal fluid returned to normal. Nine months following the onset of the eruption the neuralgia was much improved and the quadriceps muscle had returned almost to normal, with only slight persistent weakness. The right knee jerk returned, but was not quite so brisk as the left.

Case 2. A 53-year-old married woman developed typical herpes zoster on the front of the right thigh and inner aspect of the right leg. The spinal fluid showed increased protein and an increased cell count. Wasting of the quadriceps took place, with almost complete loss of power. The right knee jerk was absent. Complete flaccid paralysis of the quadriceps muscle finally took place and nocturnal neuralgia appeared. After 5 months there was marked improvement, and after 7 months almost complete recovery.

So few cases have been described that it is impossible to determine accurately the course and prognosis of paralyses complicating zoster. Recovery is slow. The increased cell count of the spinal fluid takes weeks to subside. Even after a year the neurologic findings may not reach a normal state.

A case of herpes zoster of special interest on account of the severity and wide distribution of motor and sensory disturbances has been reported by Abercrombie.[5] The patient, a man of sixty-eight, developed pain in the neighborhood of the left hip and thigh and the outer aspect of the left leg. This was accompanied by an eruption of herpetic vesicles on the left buttock and a foot drop on the left side. The herpetic eruption ran its usual course. The pain was troublesome for two months but disappeared in large part after three months. Four months following the onset of herpes zoster, a neurologic examination disclosed a generalized wasting of moderate degree of all the muscles of the left thigh and leg and extreme atrophy of the anterior-tibial group. The tibialis anticus, extensor longus digitorum and extensor proprius hallucis muscles were paralyzed, with a reaction of degeneration and resultant complete foot drop. No paresis was observed in other muscle groups. Both knee jerks were active; the left ankle jerk was diminished, and the right ankle jerk even more so, being barely perceptible. Hyperesthesia was present on the outer and anterior aspects of the left leg, and in patches along the anterior and posterior borders of this area. Hypoesthesia of higher grade, involving both pinprick and light touch, was also present on the dorsum of the left foot, extending from the ankle joint to the bases of the toes. There was no disturbance of sensation in the neighborhood of the herpetic vesicles on the left buttock. In March, 1941, one month following the first neurologic examination, a second disclosed evidence of distinct improvement.

Abercrombie in commenting on this case points out that the motor and sensory symptoms did not correspond to the distribution of peripheral nerves, and did not have the character of a root syndrome. The paralysis of the anterior tibial muscles corresponded to a lesion of the fourth and fifth lumbar segments, the sensory changes in the foot to the first and second sacral segments, and the herpetic symptoms to the third sacral segment. Taken together the features were those of a diffuse affection of the cord, almost entirely on the left side, involving both the motor and the sensory regions of the lower lumbar and the upper sacral segments and the root ganglion of the third sacral segment. Such diffuse symptoms are in accordance with the diffuse pathologic changes described by Lhermitte (quoted by Carter and Dunlop[1]).

Involvement of Special Organs

Bladder

Herpes zoster of the bladder is a rare syndrome. Rinker[35] was able to find only 4 cases in the literature. These cases have been reported by

Dubois,[36] Darget,[37] Chesterman[38] and Eisenstaedt.[39] The onset may be characterized by symptoms such as frequency, dysuria and hematuria. Both pus and blood may be found on urinalysis. Cystoscopic examination may show groups of vesicles. These vesicles sometimes ulcerate and may become covered with yellowish or grayish sloughs. They usually heal promptly with or without treatment. Lavages with 0.5 per cent silver nitrate have been recommended. Zoster vesicles may be associated with cutaneous lesions in areas such as the buttocks, or may occur without skin involvement.

Tongue

Herpes zoster of the tongue is an uncommon disease. Two cases have been reported by O'Donnell and Murphy.[40] One of these was associated with facial herpes. The first case, in a woman of fifty-five, took its onset after exposure to a cold draft. Pain appeared over the right lower jaw and the right side of the mouth. The anterior two thirds of the tongue on the right later developed a purpuric eruption covered with vesicles of pinhead size. Pain in the right ear appeared, followed by deafness. The illness had a profound constitutional effect and the patient remained in bed for a month. The second patient was a man of twenty-six. The onset took place following a cold and a typical vesicular herpetic eruption appeared on the right cheek and chin. In association with this the right anterior two thirds of the tongue showed numerous superficial ulcers, presumably ruptured vesicles. There was easily controlled pain in the right ear but no deafness. A calamine-ichthyol lotion was used on the face and permanganate mouthwashes for the tongue. Complete recovery took place in one month.

Geniculate Ganglion

The syndrome of herpes zoster oticus with facial paralysis and acoustic symptoms was first elaborated by Hunt[41] in 1907. During the thirty-five years since he crystallized medical thought concerning this syndrome, few cases have been reported, and little has been added to Hunt's original concept of the disease. He looked on herpes zoster of the face and neck as due to involvement of ganglions containing the posterior-spinal or unipolar type of cell. These cells are found in the posterior roots of the spinal cord as well as in the gasserian and geniculate ganglions. The geniculate ganglion, though located on a motor nerve, is sensory in type, having unipolar cells, and consequently is the homologue of the spinal and gasserian ganglions. With its afferent fibers, the pars intermedia of Wrisberg, and its efferent fibers, the petrosal nerves, it forms the sensory system of the facial nerve. Zoster of any zone signifies not only infection of the particular ganglion supplying this zone but also possible spread to neighboring nerves and ganglions. Geniculate herpes zoster, for example, may be associated with pain or zoster in the throat.

Hunt[42] divided geniculate herpes zoster into four main groups in accordance with severity and extent as follows: simple zoster of the external ear; simple zoster with facial palsy; simple zoster with facial palsy, accompanied by mild disturbance of the acoustic apparatus, such as diminished hearing with or without tinnitus; and simple zoster with facial palsy, together with a Ménière-like complex made up of signs and symptoms such as deafness, tinnitus, vertigo, nystagmus and nausea.

Anstie[43] in England was the first physician to report personal observations on his own subjective experiences while suffering from an attack of zoster oticus. More recently Rosenberger[44] has described his subjective experiences during an attack of zoster oticus with facial paralysis and acoustic symptoms. Both Anstie, in 1871, and Rosenberger, in 1941, misconstrued their initial symptoms, each thinking that he was developing an abscessed ear following exposure to cold. Rosenberger reports daily observations on his vestibular and auditory experiences and the associated facial paralysis. His account is well worth reading. He points out that it is probable that in many cases less severe attacks of zoster oticus manifesting only otalgia or zoster are mistakenly attributed to other causes.

Zoster and Varicella

It is now a generally accepted opinion that herpes zoster is a transmissible disease due to a filterable virus. Small epidemics have been known to occur and are said by Sutton[16] to be common. Since 1888 when Bokay suggested the possibility of a relation between herpes zoster and chicken pox, numerous examples of their association in the same family have been reported. The usual circumstances are that in a given family or closely associated group of people some children are exposed to an adult with herpes zoster. After an incubation period of fourteen to sixteen days these children come down with typical varicella without having had any known exposure to chicken pox. Epidemiologic studies of this nature have furnished substantial evidence that chicken pox may appear among persons exposed to cases of herpes zoster.[9, 45] Likewise, zoster may appear among persons exposed to chicken pox. The usual clinical observations are that, when exposed to adults with zoster, children may develop chicken

pox; and that, when exposed to children with chicken pox, adults may develop zoster. Kundratitz[46] and later Bruusgaard[11] were able to produce local and general eruptions indistinguishable from chicken pox in children by inoculating them with the fluid from zoster vesicles. Wise and Sulzberger[47] consider that zoster may be a relatively immune or allergic form of chicken pox, which occurs in those who have had previous acquaintance with the virus in the form of clinical or subclinical chicken pox. This theory would explain why as a rule herpes zoster is rare in children, chicken pox is rare in adults, inoculation of the virus from a case of chicken pox produces chicken pox in children and zoster in adults, and the zoster vesicle virus produces chicken pox in children and zoster in adults.[47]

Pathology

Carter and Dunlop[1] point out that the pathology of herpes zoster is still somewhat obscure since so few cases come to autopsy. Head and Campbell[7] in 1900 mentioned that von Bärensprung in 1861 first showed that zoster was a disease of the nervous system associated with changes in the posterior-root ganglions. They described their post-mortem findings in 21 cases in which death occurred at all stages of the disease from three days to over two years after the outbreak of the eruption, and concluded that the primary lesion was an acute round-cell infiltration with areas of hemorrhage in the affected posterior-root ganglions, and that changes found in the posterior-nerve roots and spinal cord were secondary degenerative processes. Price[48] in 1937 gave a similar account of the pathology of zoster without mentioning the existence of any spinal-cord lesions.

Such views of the pathology of herpes zoster have been challenged by the French school, notably by Lhermitte and his collaborators, who in three communications reported acute inflammatory changes not only in the ganglions but also in the posterior roots, the meninges and the cord itself. In their first case, Lhermitte and Nicholas[2] described the post-mortem findings in a patient dying seven weeks after the onset of the herpetic eruption. They found perivascular infiltration of leukocytes and mononuclear cells, with vascular dilatation and demyelination in the posterior horns of the affected segments. The anterior horns showed atrophic changes in the cells, with demyelination of the nerve roots and some lymphocytic infiltration, in spite of which no paralysis was observed clinically. There were no changes in the white matter except for one area in the pyramidal tract in which perivascular cuffing was observed. This case showed a pleocytosis of 88 lymphocytes per cubic millimeter on the second day of the eruption. Similar observations were recorded in two subsequent papers (Faure-Beaulieu and Lhermitte[49] and Lhermitte and Vermès.[50] In the former, lesions spreading to neighboring segments were found in the posterior horns. Wilson[51] records similar findings by Nicolesco, Thomas and Laminière and by Schlesinger. The view that infection involves the meninges and cord as well as the posterior root and its ganglion is supported by the frequent occurrence, according to Wilson, of a pleocytosis in the cerebrospinal fluid.

Treatment

In the management of a case of herpes zoster much can be accomplished by skillful topical applications to the skin lesions themselves. The purposes of this aspect of therapy are to protect the affected parts from irritation, friction and infection. In consideration of the evidence that the virus of zoster is present in the vesicular fluid, the use of local applications takes on an aspect of increased importance in light of the possibility that such applications might also be effective in destroying the causative organisms contained in the skin lesions.

A simple calamine lotion with phenol may be applied as a constant application to the lesions and seems to serve admirably as a soothing wash and as a means of preventing bacterial invasion of the vesicles. Vesicles so treated will usually involute at a satisfactory rate of speed, leaving no scars. The use of flexible collodion with or without antiseptic admixture may be employed as a paint or spray and has been a rather popular remedy, which also prevents secondary infection. It serves as a protective coating to lessen local irritation of the inflamed skin and may, in this way, curb the reflex arc of pain and hyperesthesia. Thick cotton pads are useful in a similar manner and seem to cut off cutaneous stimuli that would tend to aggravate hyperesthesia and pain. Dusting powders and melted paraffin are also beneficial as protective coatings to lessen local irritability and to prevent infection. Most of these topical remedies are very drying and are of chief advantage during the active vesicular stage of the disease. As the vesicles disappear and healthy, non-infected dry crusts form, it is profitable to apply boric ointment or any good ointment base such as lanolin or vaseline in order to hasten involution of the affected areas.

In many cases local therapy may be all that is needed to control discomfort. Sometimes, however, it is necessary to prescribe salicylates, phenacetin, barbiturates, codein or morphine in addi-

tion to local measures. An ethyl chloride spray over the affected dorsal-root ganglions has been found to be helpful in reducing pain. Cobra venom has been employed with prompt, complete and gratifying relief. Local anesthesia, nerve block and subcutaneous injection of thiamin chloride have been recommended as valuable pain-controlling measures. Radiant heat is comforting.

Unfiltered roentgen irradiation of the skin lesions and filtered x-ray therapy to the affected dorsal-root ganglions have been found to be valuable treatments not only in the relief of pain but also in shortening the course of the disease. X-ray used in this way has been recommended in recent treatises by leading authorities such as Pillsbury[52] and Sulzberger.[14] Keichline[53] in 1934 treated 62 cases of zoster with x-ray to the area of the eruption and to the corresponding dorsal-root ganglions, using 148 r through a 3-mm. aluminum filter at a distance of 30 cm. Ninety per cent of these patients were relieved by one dose; the others required one or two additional exposures.

Ordinary uncomplicated shingles requires little or no treatment and does not interfere with the patient's activities. Some patients, however, run a protracted and stormy course with considerable pain that is difficult to control. They require the utmost skill in management and test to the limit the physician's therapeutic legerdemain. In handling these cases it is well for the physician to be supported by a fairly complete knowledge of the modern therapeutic armamentarium pertaining to zoster. Many bizarre empirical treatments have been described, and although it is difficult to understand the manner in which they act, it is well to know about them.

Posterior-pituitary extract has been recommended by Sidlick[54] and others,[55] not only as a means of relieving pain but as a measure effective in shortening the course of the disease. Obstetric pituitrin may be administered by the subcutaneous route in doses of 0.5 to 1 cc. daily. Pituitrin is contraindicated in the presence of hypertension, coronary disease, myocardial disease and pregnancy, and it is perhaps better not to use it in elderly people even in the absence of these conditions. Portnoy[56] employed surgical pituitrin in 1 cc. doses daily by the subcutaneous route, and reported prompt relief from pain in patients whose discomfort had not been relieved by local applications, infrared heat, sodium iodide and morphine. Sulzberger and Wolf[14] recommend obstetric pituitrin in daily doses of 0.5 to 1.0 cc. for three to five days, and consider it to be the most effective form of treatment now in use. Goodman and Gilman,[57] on the other hand, state that the mechanism of action of pituitrin "is obscure and the clinical evidence of its value is contradictory."

The intravenous use of sodium iodide has been recommended by Ruggles[58] and others. Tobias[59] considers it the treatment of choice because of its effectiveness and dependability. Sulzberger and Wolf[14] consider this to be one of the two best modern forms of treatment, but warn against iodine intoxication, iodine allergy and iododerma. Ruggles advises the intravenous injection of sodium iodide in 2-gm. doses (10 cc. of a 20 per cent solution), to be given on the first, second, fourth and seventh days.

In the resistant ophthalmic type of zoster, diphtheria antitoxin has been recommended.[61] It may be given in doses of 5000 units, repeated two days later if necessary. Autohemotherapy has been in vogue for many years in the treatment of zoster. It may be given merely by removing 5 to 10 cc. of whole blood from the antecubital vein and injecting it into the gluteal region every day or two.

Cobra venom has been used extensively for the relief of pain in cancer, tabes dorsalis, Parkinson's disease[60] and painful x-ray burns.[61] During the last several years a sterile saline solution of cobra venom has been used for the relief of pain by investigators in France, India, Argentina and the United States.[62-70] Of these, Macht[67] in the United States reported 6 zoster patients who obtained striking relief. Three investigators in Italy[71] employed cobra venom successfully to relieve the pain of herpes zoster. McDowell[72] has reported its use in 6 cases, with prompt and complete relief. Both Macht and McDowell found that the pain of zoster responds much more promptly to cobra venom than it does in other conditions. No reactions either local or general were observed. No depressive reactions occurred. Very few reactions have been reported in the literature and the margin of safety seems high. For psychologic reasons it is advisable not to reveal to the patient the nature of the injections; Black[73] observed vomiting and dizziness when the patients knew the nature of the remedy, and proved conclusively that these symptoms were of psychologic origin.

Rosenak[74] reported relief from pain and regression of vesicles in zoster after intravertebral and paravertebral injections of 0.5 per cent aqueous procaine solution. Hollander[75] showed that an oil-soluble anesthetic could be injected subcutaneously into the hyperalgesic areas of zoster with prompt relief of pain, and without recurrence when the anesthesia wore off four weeks later. Secunda, Wolf and Price[76] used the method of Hollander in the treatment of 15 cases of herpes zoster, selected at random. They employed 0.5 to

2.0 per cent novocain and three preparations of anesthetic oil including Nupercain-Ciba, Hollander's formula and a modification of Hollander's formula containing benzocaine, benzyl alcohol, phenol and oil of sweet almond. The areas of hyperesthesia were mapped out by the usual methods, such as pinprick, brush and pinching. These areas were cleansed with alcohol and infiltrated subcutaneously to the point of total anesthesia. No nerve block was induced and anesthesia was confined to the area injected. Anesthesia from the novocain solution lasted one to two hours and that from the oil solution for about a week. Fourteen patients were injected with an aqueous solution of novocain and complete relief from pain ensued immediately in 13, lasting one to thirty or more hours, after which pain recurred with equal or greater severity in all but 4 patients. In 1 case the pain was made worse by the injection. Eight of the 14 patients and 1 other were subsequently treated by the injection of anesthetic oil. Complete relief of pain took place in 7 of these 9 cases. Local reactions to the anesthetic oil injections included soreness, redness, swelling and pain at the site of injection.

Relief from the acute attack was experienced by 13 of the 15 patients. Ten of these 13 were permanently relieved of all discomfort. The clinical course of the skin lesions was, however, not affected and regression of the vesicles occurred in the expected seven to fourteen days.[77] Two cases of classic post-herpetic neuralgia failed to respond to this method of treatment. Relief of pain in this series of cases was accompanied by amelioration of muscular disabilities, improved appetite, normal sleep and accelerated return to work.

The use of vitamin B_1 (thiamin chloride) in the treatment of herpes zoster is a subject of considerable general clinical interest. The fact that zoster has been reported to occur as an apparent complication of the administration of large doses of vitamin B_1 does not exclude the possibility that, in small doses, this vitamin may be of value in treating this disease. The fact that zoster occurs as a complication of the administration of large amounts of thiamin chloride provides evidence that this vitamin may have a selective action on the dorsal-root ganglions and that, in properly regulated dosage, it may be of therapeutic usefulness.

Steinberg[78] reported 3 cases of typical herpes zoster occurring as an apparent complication of the administration of a large dosage of vitamin B_1. In 1 of these cases the author was able to produce zoster on two occasions by this procedure. The other 2 patients refused further treatment on account of the zoster pain that they suffered while taking the thiamin chloride.

The case in which two attacks of zoster were produced was of special interest. The patient was a white woman of fifty-one, who was being treated for typical atrophic arthritis of twenty-six years' duration. She was given 800 units of vitamin B_1 by mouth daily and 2000 units by injection weekly. In the fourth week of this plan of treatment, typical herpes zoster developed and persisted for five weeks in spite of immediate cessation of vitamin B_1 therapy. Six weeks later the same dosage of vitamin B_1 was given again, and after the same four weeks' interval, a second typical attack of herpes zoster developed and again persisted for four weeks in spite of immediate cessation of therapy. This case is all the more remarkable because of the fact that second attacks of zoster are so exceedingly rare. Perhaps the zoster in this case was actually produced by a thiamin chloride effect on the dorsal-root ganglions and not by lowering the resistance of these ganglions to invasion by the varicella virus. Steinberg concluded that large doses of vitamin B_1 are capable of irritating peripheral-nerve plates.

The known value of thiamin chloride in the treatment of neuritis and its influence in preventing certain degenerative nerve diseases led Goodman[79] to use this vitamin in the treatment of herpes zoster. He reasoned that since the pain of zoster is due to an inflammatory process in the posterior nerve roots and to degenerative processes in the peripheral nerve tracts, perhaps thiamin chloride might be an effective form of therapy. He administered it in a series of 5 cases and obtained gratifying results in all of them. These cases were treated locally by applications of Merthiolate in collodion. Thiamin chloride was administered subcutaneously in dosage of 3000 units at varying intervals and for a varying series of injections, usually six to ten. The results were far better than those obtainable by routine local applications and salicylates. The pain was relieved more promptly and the lesions cleared at an earlier date. Total disability was noticeably decreased, and in all respects the results seemed sufficiently good to justify further investigation.

Rattner and Roll[80] used crystalline thiamin in doses of 2000 international units subcutaneously every second or third day in a series of 16 cases of herpes zoster. Seven of the patients were males and 9 were females. Two children, aged nine and thirteen, were completely relieved of symptoms after the first injection; they were treated on the third and seventh days of the disease, respectively. A third child treated on the seventh day of the disease showed no striking effect and the disease ran its usual mild course. One patient aged sixty-two obtained considerable relief following the subcutaneous injection of 6000

units on the fifth day of the disease. Another patient aged forty-eight obtained much relief after receiving 8000 units on the fifth day. In the remaining 11 cases, including 2 cases of severe post-herpetic pain, no favorable effects whatsoever were observed. The average total dose of thiamin chloride used in this series was 5620 international units. Rattner and Roll considered their results disappointing, perhaps because the patients were mostly in the older age group and perhaps because treatment may have been begun too late. Their results were quite different from the gratifying ones reported by Goodman. It may be observed that the latter used thiamin chloride in much larger total doses and by a much more persistent type of therapy.

Gordon[81] used thiamin chloride by mouth in the rather conservative dosage of 3 to 10 mg. daily in a series of 6 obstinate cases of herpes zoster of two to five months' duration. The suffering was so great in 2 cases that the patients made several attempts at suicide; all lost appetite and weight and had a reduction of hemoglobin. In all cases the zoster was thoracic in location, corresponding to the distribution of the third to twelfth spinal segments, involving in each case two or three segments of the spinal cord. Neurologic changes, such as abolition of the patellar and Achilles reflexes, anesthesia, hypesthesia and hyperesthesia, were present in some cases. Gordon found in the treatment of these rather severe cases that vitamin B_1 alone was no more effective than the customary standard forms of treatment. He found, however, that when he combined vitamin B_1 with sedatives, local applications containing cocaine or procaine, applications of heat and cold and autohemotherapy, he was able to produce striking improvement of the pain and skin lesions.

In the papers of Gordon and of Rattner and Roll may be found extensive reviews of the literature on the use of thiamin chloride in the treatment of numerous and varied diseases, including neuritis, sciatica, general debility, inflammatory and degenerative diseases of the central nervous system, multiple sclerosis, degeneration of the pyramidal tracts, beriberi, pruritus ani, pruritus vulvae, inoperable cancer, diabetic polyneuritis, alcoholic neuritis, colitis, irradiation illness and acrodynia in children. These reports, all written in a rather optimistic vein, made it seem rational to use thiamin chloride in the treatment of zoster.

Smith[82] has given a helpful analysis of the extensive literature on the treatment of herpes zoster, and has reported the excellent results that he obtained by the extraordinary procedure of injecting thiamin chloride into and beneath the skin of areas involved in the skin eruption of herpes zoster. Three patients were treated by this method and all obtained prompt and remarkable relief. Only relatively small areas were treated, and therefore it seemed unlikely that a purely local effect on nerve endings could explain the results. Smith considered that absorption along the lymphatics might bring about a high concentration of thiamin chloride in the region of the affected dorsal-root ganglions. He used a combination of intradermal and subcutaneous injections of thiamin chloride daily for a series of three or four injections. Striking relief of discomfort usually took place within a few hours following the first injection. Complete and permanent relief ensued after the third or fourth injection.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 29181

PRESENTATION OF CASE

A fifty-eight-year-old physician entered the hospital because of intermittent attacks of low-back pain.

Eleven years prior to admission, after a strenuous day of ice skating, the patient developed severe localized low-back pain that interfered with his activities for three days but did not confine him to bed. Six years later a similar attack of pain occurred after ice skating and lasted three days. During these episodes he was unable to dress without assistance or bend forward because of the pain, and once, while shaving, his legs gave way and he fell to his knees, but soon recovered. Five or six times during the next four years he was awakened from a sound sleep by severe "gripping" pain that seemed to be just above the rectum and was relieved by warm-water enemas. Five months before entry the patient developed a feeling of tension in the posterior thigh muscles. Two weeks before admission, steady, sharp pain developed in the region of the lower lumbar spines that radiated down the posterior aspects of both thighs to the knees and bilaterally to the inguinal regions and scrotum. The lower-lumbar and posterior-thigh pain was intensified by coughing, sneezing or straining at stool. When sitting, "pins and needles" developed in the pressure areas of the buttocks. One week before entry the patient noticed numbness on the outer aspect of the left foot, which seemed to disappear after a night's rest. Five days before admission, while applying a cast, excruciating pain started in the lower lumbar region and radiated down the posterior aspects of both legs to the heels. There was a sensation of numbness and of "pins and needles" in both feet, and the patient was unable to walk. After forty minutes' rest the pain gradually subsided. During the night he was awakened by a similar attack, which required morphine for relief. Nine hours later he found that he was unable to void and had developed anesthesia involving the penis, scrotum, perineum and perianal region. During the next day the anesthesia diminished rapidly in intensity but the patient had to be catheterized. After that he was able to void spontaneously. His bowels had not moved for five days, although he had previously been regular. Adhesive-tape leg traction was applied at a community hospital, after which the patient was quite comfortable. A lumbar puncture demonstrated complete block and yielded only a few cubic centimeters of yellow fluid, which had a protein content of 750 mg. per 100 cc. The exact location of this puncture was not known.

Physical examination revealed a well-developed and well-nourished man lying comfortably in bed. The heart, lungs and abdomen were normal. There was weakness of dorsiflexion of the right foot and great toe. The strength of the other muscle groups on this side could not be determined because of the interfering pain in the lumbar region, but a full range of motion seemed present except for straight leg raising, which was limited on the right to 45° and on the left to 30° by sharp pain in the posterior aspect of the thigh and legs. There was slight tenderness to the right of the fourth and fifth lumbar spinous processes. The knee jerks were present and equal. The ankle jerk was absent on the right, but normal on the left. There was impairment of all forms of sensibility over the medial parts of the buttocks, in the perineum and over the lateral half of the leg, dorsum and sole of the foot on the right, with marked subjective numbness of the third, fourth and fifth toes bilaterally, which was more noticeable on the right. The neurologic examination was otherwise not remarkable. The patient reported loss of sphincter sensation during defecation.

The blood pressure was 155 systolic, 85 diastolic. The temperature, pulse and respirations were normal.

Examination of the blood revealed a red-cell count of 5,900,000, with a photo-electric hemoglobin of 16.6 gm. per 100 cc., and a white-cell count of 6600, with 68 per cent neutrophils, 16 per cent lymphocytes, 11 per cent monocytes, 3 per cent eosinophils and 2 per cent basophils. The urine was acid and had a specific gravity of 1.020, and the sediment contained 2 red cells and 5 white cells per high-power field. A blood Hinton test was negative. A lumbar puncture in the interspace between the first and the second lumbar vertebra revealed normal dynamics. The fluid was slightly xanthochromic, with a protein of 266 mg. per 100 cc. and was otherwise negative.

A roentgenogram of the chest was normal. X-ray films of the spine and pelvis disclosed calcified deposits along the anterior margins of the spine in the region of the interspinous ligaments. In the lower dorsal and upper lumbar regions there were small spurs on the anterior margin of the bodies of the vertebras. The intervertebral

spaces were not narrowed or deformed. There were no localized widenings of the neural canals or erosion of the lateral masses. Injection of iodized oil revealed a block at the level of the fourth lumbar disk (Fig. 1). There was a round-

FIGURE 1. *Iodized Oil Arrested above the Fourth Lumbar Intervertebral Disk.*

ed filling defect above the inferior margin of the lamina. The nerve roots were displaced medially, and the defect was more pronounced anteriorly and on the right side.

An operation was performed on the fourth hospital day.

DIFFERENTIAL DIAGNOSIS

DR. JOST MICHELSEN: After reading this story one can reach a preliminary conclusion that ought to prove correct. This patient had a surgical lesion in the lower lumbar spine. We may further assume that the surgeon expected to find a herniation of the nucleus pulposus at the fourth lumbar interspace and that he also was prepared to open the dura and explore the cauda equina if no extradural lesion was found.

The indications for surgical exploration are obvious: there was a block on lipiodol examination; there was also dynamic block, with a protein of 750 mg. per 100 cc., below the lesion; the dynamics were normal above the lesion, where the protein was lower but still abnormally high.

Exaggeration of pain on coughing, sneezing and straining is further evidence of a space-limiting lesion in the spinal canal, which, however, is of minor significance in view of the lumbar-puncture and lipiodol findings.

We also know that the upper level of this lesion was at the fourth lumbar disk and that the mass producing the defect was anterior and on the right side. The defect was rounded, and apparently there was no cap formation, which is characteristic of an intradural lesion. All these observations are suggestive of a large herniation of the nucleus pulposus. So are the history and findings, although they do not correspond entirely with those obtained in the average case. Consistent are the long story with intermittence of symptoms, the limitation of forward bending, and the fact that the low-back pain — the only symptom for years — preceded the onset of radiating pain in the lower extremities and the other symptoms, which will be discussed later. Perhaps it is also sigr.ificant that the two first attacks of back pain started after ice skating.

The distribution of the nocturnal pains just above the rectum is uncommon, and I also do not quite understand how they were relieved by warm-water enemas, whatever their nature might have been. Fortunately the sharp pains developing two weeks before entry were again in a more appropriate place, but it should also be noted that there was radiation of this pain down the posterior aspects of both legs and to both inguinal regions as well as to the scrotum. Pain in the inguinal region from a lesion whose upper level has been demonstrated to be at the fourth lumbar disk must be referred pain. Subjective sensory disorders at segments higher than the actual lesion have been observed in ruptured disks of the lumbar as well as the cervical spine; one wonders if perhaps they are due to the peculiar setup of the lumbar and brachial plexuses.

An outstanding feature of these sharp excruciating pains is their bilateral involvement of the lower extremities. The bilateralness is further emphasized by paresthesias in both feet, weakness of the bladder and bowel, and sensory disturbances in the saddle area. The findings on examination are in close agreement with these symptoms. There was saddle anesthesia for all forms of sensation, that is, in the area supplied by the third to fifth sacral segments, as well as numbness of the third, fourth and fifth toes bilaterally or in the first and second sacral dermatomes. On the right leg the anesthesia apparently had a first and second sacral distribution. Also recorded is weakness of the anterior tibialis and extensor hal-

lucis longus muscles, disclosing disease of the fifth lumbar and first sacral anterior roots. In other words, the posterior roots of the cauda equina from the fifth lumbar to the fifth sacral on both sides and the fifth lumbar and first sacral anterior roots on the right were damaged.

It is known that a large herniation of a nucleus pulposus can produce the symptoms that this patient had, although the commoner syndrome consists of low-back pain, tenderness of the lower lumbar spaces, sciatic pain on one side, well-localized paresthesia and hypesthesia, limitation of straight leg raising, diminution of the ankle jerk, increased protein in the cerebrospinal fluid, a lipiodol defect and intermittence and recurrence of symptoms and signs.

I therefore come to the conclusion that the surgeon found a large herniation of the fourth lumbar disk on the right side extending far toward and beyond the midline. This is the commonest extradural lesion in the lumbar area, and there is no evidence, so far as I can see, to indicate any other type of extradural lesion. I consider the possibility of an intradural mass as remote in view of the course of the disease.

DR. RICHARD SCHATZKI: The lumbar spine shows no evidence of disease, as stated in the report, aside from minor degenerative changes. Lipiodol shows a complete block just above the lower edge of the fourth lumbar vertebra. The lipiodol runs down to that point and stops there. A little lipiodol runs out into the nerve sheaths.

You can see that the defect is larger on the right side than on the left, but complete block is present. The lateral view shows that the defect is definitely larger anteriorly. There is not the real cap formation that one usually sees in patients with a well-rounded tumor in this area, such as a neurofibroma. In any tumor, whether intradural or extradural, its smooth surface is demonstrated by the lipiodol, and is usually more symmetrical with an intradural tumor than with an extradural one.

Do you want me to go beyond that?

DR. CHARLES S. KUBIK: Have you a film more characteristic of ruptured disk?

DR. SCHATZKI: Arrest of the lipiodol is by no means the usual finding in cases of ruptured disk. Here, in another case, you see the characteristic appearance of the lipiodol. This patient had a rupture of the fourth disk on the left side, which produced a localized defect of the lipiodol column opposite the disk that, as Hampton and Robinson* reported, has the shape of an anvil or half an anvil, with smooth superior and medial surfaces.

DR. JAMES B. AYER: I think it interesting that the patient in the present case had a referred type of sensory disturbance well above the involved area. This was largely a paresthesia, which does happen.

DR. W. JASON MIXTER: Pain in the groins is not too uncommon. You see that every once in a while with a lesion of the fourth or fifth lumbar disk.

*Hampton, A. O., and Robinson, J. M. Roentgenographic demonstration of rupture of intervertebral disc into spinal canal after injection of lipiodol with special reference to unilateral lumbar lesions accompanied by low back pain with "sciatic" radiation. *Am. J. Roentgenol.* 36:782-803, 1936.

CLINICAL DIAGNOSIS

Ruptured intervertebral disk (fourth lumbar), with compression of nerve roots.

DR. MICHELSEN'S DIAGNOSIS

Ruptured intervertebral disk (fourth lumbar).

ANATOMICAL DIAGNOSIS

Ruptured intervertebral disk (fourth lumbar).

PATHOLOGICAL DISCUSSION

DR. KUBIK: A large protrusion of the nucleus pulposus of the fourth lumbar disk on the right side was found at operation. The spine was not fused. Three months later the patient was back at work, taking care of a busy practice.

CASE 29182

PRESENTATION OF CASE

A sixty-four-year-old salesman entered the hospital because of weakness, fainting spells and tarry stools.

The patient apparently was quite well until nine months before admission when at a community hospital a transurethral resection of the prostate was performed because of frequency, urgency, nocturia and a high bladder residual. Soon after leaving the hospital he frequently noticed that his stools were tarry. Three months before entry the patient suddenly felt weak, cold and sweaty and fainted. He recovered after several minutes, but felt exceedingly weak for a day. Because he could not carry on his daily duties, he took a vacation. A month before admission he had several similar fainting spells but quickly recovered consciousness spontaneously. Several hours later he usually passed a semiliquid stool that resembled "coffee-grounds." His physician prescribed bed rest and an ulcer diet amplified with iron, but this failed to relieve the persistent weakness and easy fatigability. There was no nausea, vomiting, pain, constipation, jaundice or persistent diarrhea. The patient had lost 10 pounds during the period of illness.

The family and past histories were noncontributory.

Physical examination disclosed a pale, tired-looking man who lay quietly in bed. A soft systolic murmur was audible over the entire precordium. The lungs and abdomen were normal.

The blood pressure was 118 systolic, 70 dias-

tolic. The temperature, pulse and respirations were normal.

The red-cell count was 3,870,000, with a photoelectric hemoglobin of 9.5 gm. per 100 cc., and the white-cell count 11,200. The urine was negative. A blood Hinton test was negative. The nonprotein nitrogen was 24 mg., and the protein 6.3 gm. per 100 cc.; the chloride was 97.6 milliequiv. per liter. The prothrombin time was 21 seconds (normal, 16 seconds).

A barium meal showed curling in the lower half of the esophagus. There was a slight hiatus hernia. The stomach showed a filling defect, measuring 6.0 by 4.3 cm., on the anterior wall in the region of the angulus and lying toward the lesser curvature. This was clearly visible only with the patient lying prone. The lesion had a broad shape and extended upward to the level of the esophageal hiatus but lay anterior to it and had a lobulated surface. There was an ulceration on the lesser curvature, and in the films brought in by the patient the filling defect appeared to extend upward to the region of the cardia. The mucosa on the posterior wall of the stomach appeared normal. The duodenum and proximal jejunum were normal.

A roentgenogram of the chest showed no definite evidence of metastases. There was some increased density in the right second interspace consistent with old infection. The apex of the heart was somewhat rounded, and the aorta was slightly tortuous.

On the eighth hospital day an operation was performed.

Differential Diagnosis

Dr. Edward Hamlin, Jr.: This case resolves itself into the discussion of tumors of the stomach, and in large part the answer should be given by the x-ray men. I might say that I do not know anything about the x-ray interpretation of tumors of the stomach.

We may also consider the case from a statistical point of view. Of roughly 5000 surgical specimens of stomachs resected during a period of years at the Mayo Clinic,* about 2300, or nearly half, were cancers; about half that number were peptic ulcers; lymphosarcomas numbered 35; there were 12 fibrosarcomas or leiomyosarcomas, 2 angiosarcomas, and 4 cases of tuberculosis, with no cases of syphilis. Considering the present case from more than a statistical or an x-ray standpoint does not seem to be possible.

The predominant feature of the history of this man was bleeding. Of the lesions in the stomach that are more prone to bleed than others I should think lymphosarcoma was probably as prominent as any. The x-ray appearance described as a lobulated surface does not seem applicable to carcinoma.

This might be a good time to look at the x-ray films, except that I do not see how I can get any more information without asking direct leading questions.

Dr. Richard Schatzki: I do not know the answer, so you may question me all you wish.

These are the films brought in from the outside. They show this large filling defect (Fig. 1). The lower edge is clearly indicated about 7 cm. proximal to the pylorus, and is markedly lobulated. There is also a deep, rather linear, crater somewhere near the center of the defect. The upper edge is not so clearly visible, but I doubt if it goes to the cardia. These are the films taken here. You see in the big films the same appearance that was seen on the outside films, except that the crater is not filled so much.

Dr. Hamlin: These films certainly are not spectacular.

Dr. Schatzki: Here are two spot films. The lobulation on one of them is by no means so striking as that on the big films, and I am inclined to think that that is of some significance.

Dr. Hamlin: One thing that I do remember about x-ray interpretation of tumors of the stomach, which may not be of significance here, is that some leiomyomas of the stomach have a very characteristic picture, which resembles in outline that of the human cervix. Purely on that basis, although I have no business making an interpretation on x-ray evidence, I will make leiomyoma or leiomyosarcoma my first choice.

Dr. Schatzki: The differential diagnosis of a case like this lies between the three tumors that Dr. Hamlin has mentioned. Lymphoma is the least likely and the argument usually centers around a spindle-cell tumor or just another cancer, and at times it is impossible to make a differential diagnosis, since carcinoma may imitate the x-ray appearance of a spindle-cell sarcoma. The central ulceration that is typical of a sarcoma may be just a cleft in the cancer that has filled with barium. On the other hand, this sharp edge is very unusual in cancer. It occurs occasionally if there is a dish-like cancer that has an elevated "life-saver" rim with a smooth edge. Such a cancer, however, has a large central crater, whereas this is relatively small. I think I shall agree with Dr. Hamlin and call it an intramural spindle-cell tumor that has become ulcerated.

Dr. Tracy B. Mallory: Benign or malignant?

Dr. Schatzki: It is impossible to say on the x-ray evidence.

Dr. Mallory: This is essentially an x-ray diagnosis. Has Dr. Sosman an opinion?

*Eusterman, G. B., and Balfour, D. C. *Stomach and Duodenum*. 958 pp. Philadelphia: W. B. Saunders Company, 1935. P. 73.

Dr. Merrill C. Sosman: It is interesting that the films with the patient prone show an irregular lobular outline. The spot film shows a smooth outline when the patient was upright or more probably supine. So probably the prone film gives a truer appearance of the edge of the tumor, and working on the same hypothesis or line of reasoning as Dr. Schatzki used, I should say that it fits better with adenocarcinoma and that the cleft or fissure is merely barium that came in between the two lobules of adenocarcinoma. Just to be different, I shall put my money on that horse.

Figure 1. *First Film of Stomach Showing Filling Defect with Lobulated Border and Central Crater.*

Clinical Diagnosis

Leiomyoma of stomach.

Dr. Hamlin's Diagnosis

Leiomyoma or leiomyosarcoma of stomach.

Anatomical Diagnosis

Fibrosarcoma of stomach.

Pathological Discussion

Dr. Mallory: On exploration a smooth, movable tumor was found, which was nearly spherical. On palpation of the external surface of the stomach, Dr. A. W. Allen thought it was a spindle-cell tumor. He therefore did a relatively limited gastric resection and provided us with this specimen. You can see that the mucosa over the entire tumor is intact except for a small area of ulceration that is hardly more than a dimple on the top of the dome. A fistulous tract only a couple of millimeters in diameter extends downward from the ulcer to a cavity perhaps 2 cm. in diameter that lies deep in the center of the tumor. Obviously barium would have had some difficulty in penetrating to this central cavity.

Dr. Schatzki: It probably was filled in the outside films but not in the hospital ones.

Dr. Mallory: On microscopic examination the tumor proved to be a fairly rapidly growing, poorly differentiated, spindle-cell sarcoma, which was undoubtedly malignant and probably fibrosarcomatous.

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The Journal does not hold itself responsible for statements made by any contributor.

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ANNUAL MEETING

The one hundred and sixty-second anniversary of the Massachusetts Medical Society will be held at the Hotel Statler, Boston, on May 24, 25 and 26; the program of the meeting appears elsewhere in this issue of the *Journal*.

According to the schedule adopted in 1942, the annual meetings of the supervising censors and of the Council will be held in the late afternoon and evening, respectively, of Monday, May 24, the day before the opening of the regular sessions.

The Committee on Arrangements has prepared an extremely timely, interesting and valuable program of scientific papers. As would be expected, the majority of these are related, directly or indirectly, to the war effort.

The first morning will be devoted to a symposium on burns, with special reference to the Cocoanut Grove disaster. Following the business portion of the annual meeting of the Society, Dr. Edward P. Bagg, of Holyoke, will deliver the annual oration. The afternoon session largely concerns wartime medical topics, and after the annual dinner that evening, at which the guest speaker will be Mr. James A. Hamilton, president of the American Hospital Association, the Shattuck Lecture will be given by Dr. George W. Thorn, the newly appointed Hersey Professor of the Theory and Practice of Physic at the Harvard Medical School.

The program for the second day covers a miscellaneous group of topics. The following papers should be of particular interest: "Newer Chemotherapeutic Agents," by Dr. Charles A. Janeway; "Meeting Nutritional Requirements in Time of War," by Dr. Russell M. Wilder, of Washington, D. C.; "Oxygen Therapy and Positive Pressure as Related to Gas Poisoning in War and in Civilian Disorders," by Dr. Alvan L. Barach, of New York City; and "Present-Day Rapid Methods of Treating Syphilis," by Dr. Harold N. Cole, of Cleveland. The various sections will hold luncheon meetings, and at each a short talk is scheduled.

The scientific and technical exhibits, although somewhat smaller than they have been in the last few years, will be no less interesting. And, as in previous years, there will be a continuous motion-picture program on both days.

Two of the usual accompanying activities — the ladies' entertainment and the golf tournament — have been canceled, owing to the difficulties of transportation.

The Committee on Arrangements, which is composed of Dr. Gordon M. Morrison, chairman, and Drs. Roy J. Heffernan, Sidney C. Wiggin, Richard I. Smith and G. Guy Bailey, deserves great praise for planning such an attractive program

at a time when it is difficult to obtain authoritative speakers. This can be evidenced only by a good attendance of the members of the Society, and all are urged to be present.

QUOTAS FOR MEDICAL OFFICERS

To determine how many of a given number of medical officers should be obtained from each state and how these needs should be allocated to subdivisions within the state seems to be a simple matter. On the basis of percentages, knowing the total number of men required and the numbers of licensed physicians in the country as a whole, in the state and in the subdivision, figures can be jotted down within a few minutes, particularly if one is adept in the use of a slide rule. But the matter of determining quotas is far more difficult.

Physicians are not divided throughout the country according to population. They tend to congregate in cities, especially large cities, where opportunities are greater than they are in the rural districts. There, modern hospital facilities are available, the need for specialists, including those concerned with industrial medicine, and for instructors and research workers, is great, the chances for the continuous postgraduate education that is so necessary for the successful practice of medicine are excellent, and life, by and large, is relatively easy. As a result the number of physicians per unit population is higher in Massachusetts than in Kansas, and higher in Suffolk County of Massachusetts than in Franklin County. As a corollary it is obvious that to cut the number of physicians in half would create more of hardship on the civilian population in Kansas than it would in Massachusetts, and more in Franklin County than in Suffolk County. Thus, the percentages of physicians from the manufacturing states and from the large cities must be higher than those from the agricultural states and the rural communities. On the other hand, the strict application of the one-physician-per-fifteen-hundred-inhabitants yardstick to large cities is impracticable, owing to the nature of local medical practice. In addition, it must be acknowledged that, generally speaking, a commission in the Army or Navy offers more financial security to the physician in the country than it does to his counterpart in the city. All these facts — and many more — have been considered by the Directing Board of the Procurement and Assignment Service in Washington and by the state committees of this agency when quotas and allocations were determined.

The total quota for Massachusetts — 2400 medical officers — is high on a percentage basis, as are the allocations to the districts comprising Metropolitan Boston. However, in spite of statements to the contrary, the medical profession has responded wholeheartedly to the call, and according to Dr. Reginald Fitz, state chairman of the Procurement and Assignment Service, close to the desired number of men have already been obtained. This does not mean that no more are needed, but it does signify that those physicians who volunteer in the future must, for the most part, come from the urban districts.

MEDICAL EPONYM

Valsalva Maneuver

This maneuver was described by Antonio Maria Valsalva (1666–1723) in his book *De Aure Humana Tractatus* [*Treatise on the Human Ear*] (Utrecht, 1717). A portion of the translation from page 84 of a later edition (Leyden, 1735) is as follows:

> If a person who has a continuously discharging abscess in or near the eardrum attempts, with his mouth and nostrils closed, to compress the air inside, then, as this is done, the bloody matter usually flows out freely into the auditory meatus.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

COMMITTEE ON MATERNAL HEALTH

Obstetric Case History: Acute appendicitis complicating labor

A twenty-four-year-old primipara was first seen when approximately thirty-five weeks pregnant. Her past history was essentially negative. She had had no contagious diseases, nor had she been operated on. Catamenia began at the age of twelve and had always been regular, lasting three days. The early part of the pregnancy was accompanied by a normal amount of morning sickness and some frequency of urination. When about twelve weeks pregnant, she was said to have had an attack of abdominal pain with vomiting. A similar attack occurred two months later. Except for these incidents, the pregnancy had been uneventful.

Physical examination showed a well-developed and well-nourished woman. The heart and lungs were negative. The breasts were consistent with pregnancy. The blood pressure was 120 systolic, 80 diastolic. The uterus was enlarged to the size of a pregnancy of approximately eight months. The fetal heart was heard, and the external pelvic measurements were within normal limits.

Five days after this first office visit, the patient was having a good deal of pain in the upper part of the abdomen. Physical examination revealed no abdominal spasm, and the temperature was normal. This distress was relieved by an enema.

The next evening the membranes ruptured spontaneously, and the patient began to have cramp-like abdominal pains every twenty minutes. Twelve hours later she had a temperature of 100°F., a pulse of 80, and a white-cell count of 17,000. She was definitely in labor. Rectal examination revealed an obliterated cervix and a head that was well descended into the pelvis; the cervix was dilated to admit two fingers. The patient was lying in bed comfortably. Her tongue was dry. The left side of the abdomen was soft, but the right side was tender and spastic. This tenderness did not extend into the pelvis. A catheter specimen of urine showed no evidence of pyelitis. The elevated white-cell count, the moderate rise in temperature and the rigidity and tenderness of the abdomen pointed to an acute abdominal condition, the most likely diagnosis being acute appendicitis. A surgical consultant confirmed the diagnosis of an acute condition of the abdomen, and an obstetric consultant advised immediate operation.

Under spinal anesthesia the abdomen was opened by a right paramedian incision. Free purulent fluid was found in the right lower quadrant. The appendix, which was lying behind the uterus, covered with a layer of fibrin and pus exuding from its tip, was removed. The stump was not inverted. Ten grams of sulfanilamide was put in the abdominal cavity, and the abdominal wall was closed in layers, with a drain to the site of the appendix.

Labor proceeded during the day, and a simple forceps delivery was accomplished about ten hours after the operation. Spinal anesthesia was again used. The baby cried lustily. It weighed 5 pounds, 9 ounces, at birth and was discharged well from the hospital.

For the first three days the patient's convalescence was accompanied by a temperature ranging from 98 to 101°F. and a pulse varying from 100 to 120. There was no postoperative vomiting, and little distention. Nine days after the operation the patient complained of pain in the right shoulder. The temperature rose to 100°F., and the white-cell count was 9000; x-ray examination showed an infarct in the right lung. The temperature remained elevated for forty-eight hours, but there was no cough. The drain and stitches were removed on the thirteenth postoperative day. Subsequent convalescence was uneventful.

The pathological report was "acute appendicitis."

Comment. This case illustrates the intelligent handling of acute appendicitis during labor, which is relatively uncommon. If operation had been delayed until after the baby had been delivered, it is quite likely that a fatal peritonitis would have developed. When this case was seen, it was evident that an acute condition of the abdomen existed. Pyelitis was eliminated by negative urinary findings. A pedunculated fibroid and an ovarian cyst with a twisted pedicle were considered, but no definite tumor could be demonstrated. Hence, the diagnosis of appendicitis seemed to be the logical one.

Any acute abdominal condition during pregnancy demands surgery, and the sooner the operation is performed, the better the chance for recovery, even if the patient is in labor.

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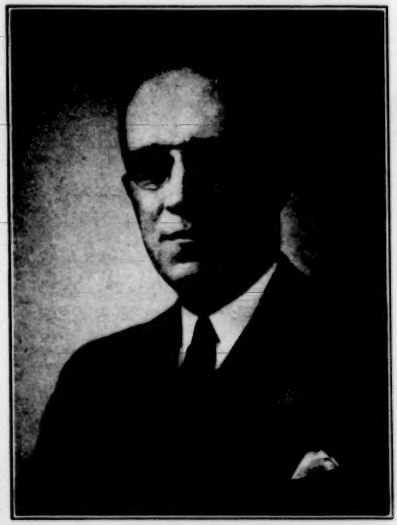

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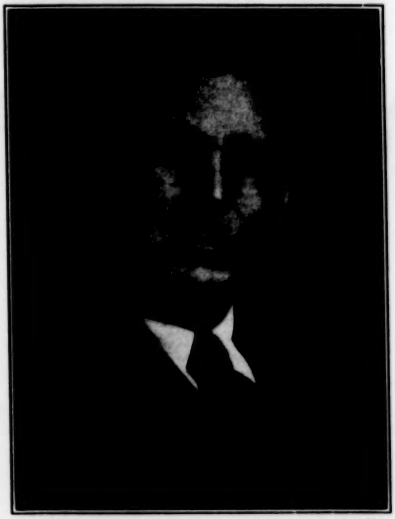

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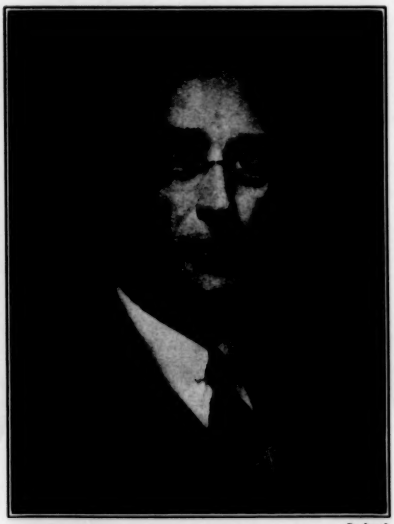

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PROGRAM OF THE ONE HUNDRED AND SIXTY-SECOND ANNIVERSARY OF THE MASSACHUSETTS MEDICAL SOCIETY

Monday, Tuesday and Wednesday, May 24, 25 and 26, Hotel Statler, Boston

THE Registration Desk will be located on the Mezzanine Floor, and all who attend the meeting are requested to register.

Owing to the lack of personnel and food supplies, the Hotel Statler must be informed concerning the number of members who will attend the various luncheons and dinners. Attendance is not limited, but tickets *must* be obtained in advance. It is doubtful whether any tickets will be available at the time of the meeting.

MONDAY EVENING, MAY 24

Annual Meeting of the Council of the Massachusetts Medical Society

5:00 **Supervising Censors' Meeting** (PARLOR D).

6:00 **Cotting Supper** (SALLE MODERNE).

7:00 **Council — Annual Meeting** (GEORGIAN ROOM).

TUESDAY MORNING, MAY 25

First General Session

GEORGIAN ROOM

Dr. Peirce H. Leavitt, *Chairman*
Dr. Leroy E. Parkins, *Co-chairman*

Symposium: Burns, with Special Reference to the Cocoanut Grove Disaster

9:00 *Burn Shock and Its Treatment.* Dr. Charles C. Lund: visiting surgeon, Boston City Hospital, and assistant professor of surgery, Harvard Medical School.

9:15 *Surface Treatment.* Dr. Oliver Cope: associate visiting surgeon, Massachusetts General Hospital, and assistant professor of surgery, Harvard Medical School.

9:30 *Chemotherapy.* Dr. Champ Lyons: assistant surgeon, Massachusetts General Hospital, and instructor in bacteriology and associate in surgery, Harvard Medical School.

9:45 *Respiratory Complications.* Dr. Maxwell Finland: chief, Fourth Medical Service, and associate physician, Thorndike Memorial Laboratory, Boston City Hospital, and assistant professor of medicine, Harvard Medical School.

10:00 *Pathology of Respiratory Burns.* Dr. Timothy Leary: medical examiner, Suffolk County, Southern Division.

10:15 *Medical-Examiner Service at the Disaster.* Dr. William J. Brickley: medical examiner, Suffolk County, Northern Division.

10:30 *Discussion.*

11:00 **Annual Meeting of the Massachusetts Medical Society.**

GEORGIAN ROOM

Annual Oration (following business meeting): *Small Puddles.* Dr. Edward P. Bagg, Holyoke: chief medical evacuation officer, Region II; president, staff of Holyoke Hospital.

Annual Luncheon (PARLORS A, B AND C): tickets MUST be procured in advance of the meeting.

TUESDAY AFTERNOON, MAY 25

Second General Session

GEORGIAN ROOM

Dr. Roger I. Lee, *Chairman*
Dr. Dwight O'Hara, *Co-chairman*

2:00 *The Medical Administration of Selective Service within a State.* Lt. Col. Victor D. Washburn, M.C., A.U.S.: state medical officer, Selective Service, Massachusetts.

2:20 *Current Considerations of the Army Anesthesiologist.* Major Stevens J. Martin, M.C., A.U.S., Fort Dix, New Jersey: chief of Section of Anesthesia and Section of Operating Pavilion Resuscitation and Oxygen Therapy, Tilton General Hospital.

2:40 *Cervical Spinal-Cord Injuries: A study of 101 cases.* Dr. Donald Munro: surgeon-in-chief and head of the Department of Neurological Surgery, Boston City Hospital.

3:00 *Wartime Responsibilities of the Public Health Service.* Dr. Warren F. Draper, Washington, D. C.: Acting Surgeon General, United States Public Health Service.

3:20 *Global Malaria.* Brig.-Gen. James S. Simmons, M.C., U.S.A., Washington, D. C.: director, Preventive Medicine Division, Office of the Surgeon General.

3:40 *The Increased Danger from Tropical Disease in the Present War.* Col. Richard P. Strong, M.C., A.U.S., Washington, D. C.: director, Division of Tropical Medicine, Army Medical School.

4:00 *How to Improve Fracture Results.* Dr. Charles L. Scudder: consulting surgeon, Massachusetts General Hospital.

4:20 *Diverticulitis of Colon: Its importance in general practice and its treatment.* Dr. Edward L. Young: surgeon-in-chief, Faulkner Hospital, and visiting surgeon, Massachusetts General Hospital; and Lieut. Edward L. Young, 3d, M.C., A.U.S., Boston.

4:40 *Therapy of Hypertension.* Dr. Robert W. Buck: visiting physician, Pratt Diagnostic Hospital, and chief, Nephritic Clinic, Boston Dispensary.

TUESDAY EVENING, MAY 25

7:00 **Annual Dinner of the Massachusetts Medical Society.**

GEORGIAN ROOM

Tickets for the dinner MUST be procured in advance.

Presiding: Dr. George Leonard Schadt, president, Massachusetts Medical Society.

Guest speaker:

Mr. James A. Hamilton, New Haven, Connecticut: president, American Hospital Association; director, New Haven Hospital, and professor of hospital administration, Yale University.

The Shattuck Lecture (SALLE MODERNE) (following annual dinner). *Physiological Considerations in the Treatment of Nephritis.* Dr. George W. Thorn: Hersey Professor of the Theory and Practice of Physic, Harvard Medical School, and physician-in-chief, Peter Bent Brigham Hospital.

WEDNESDAY MORNING, MAY 26

Third General Session

GEORGIAN ROOM

Dr. James W. Manary, *Chairman*
Dr. Edward J. O'Brien, Jr., *Co-chairman*

9:00 *Newer Chemotherapeutic Agents.* Dr. Charles A. Janeway: assistant professor of pediatrics, Harvard Medical School; visiting physician, Children's Hospital.

9:20 *Modern Pharmacy and the Medical Profession.* Mr. Howard C. Newton: president, American Association of Colleges of Pharmacy, and dean, Massachusetts College of Pharmacy.

9:40 *The Work of the Boards of Registration in Medicine and Nursing.* Dr. H. Quimby Gallupe: secretary, Massachusetts Board of Registration in Medicine, and secretary, Massachusetts Board of Registration in Nursing.

10:00 *The Use of Refrigeration in Peripheral Vascular Disease.* Dr. E. Everett O'Neil: visiting surgeon, Boston City Hospital, and clinical professor of surgery, Boston University School of Medicine.

10:20 *Newer Concepts of Gonorrhea.* Dr. Samuel N. Vose: associate professor of genitourinary surgery, Boston University School of Medicine.

10:40 *Chronic Adhesive Subacromial Bursitis ("Frozen Shoulder").* Dr. Gilbert E. Haggart: attending orthopedic surgeon, New England Baptist and Deaconess hospitals, and chief of the Bone and Joint Service, Lahey Clinic.

11:00 *Physical Therapy in Arthritis.* Dr. William B. Snow, New York City: associate in medicine, College of Physicians and Surgeons, Columbia University, and director of physical therapy, Medical Center (Presbyterian and allied hospitals).

11:20 *Meeting Nutritional Requirements in Time of War.* Dr. Russell M. Wilder, Washington, D. C.: chief, Civilian Food Requirements Branch, United States Department of Agriculture.

11:40 *Oxygen Therapy and Positive Pressure as Related to Gas Poisoning in War and in Civilian Disorders.* Dr. Alvan L. Barach, New York City: associate professor of clinical medicine, College of Physicians and Surgeons, Columbia University.

WEDNESDAY NOON, MAY 26

Section Meetings and Luncheons

12:00 m.–2:00 p.m.

Tickets for the luncheons MUST be procured in advance.

Section of Medicine

PARLORS B AND C

Dr. Laurence D. Chapin, Springfield, *Chairman*
Major Richard P. Stetson, M.C., A.U.S., *Secretary*

The Problem of Civilian Medical Practice in Wartime. Dr. John J. Dumphy, Worcester.

Section of Surgery

PARLOR A

Dr. James C. McCann, Worcester, *Chairman*
Dr. Oliver Cope, Cambridge, *Secretary*

Surgical Lessons of the Pearl Harbor Attack. Dr. John J. Moorhead, New York City: professor of surgery, New York Postgraduate Medical School and Hospital, and director, Department of Traumatic Surgery (retired).

Section of Pediatrics

THE JUNIOR LEAGUE, ZERO MARLBOROUGH STREET

Dr. James M. Baty, Belmont and Brookline, *Chairman*
Dr. Gerald N. Hoeffel, Cambridge, *Secretary*

Nephritis in Childhood. Dr. Allan M. Butler.

Section of Obstetrics and Gynecology

WOMEN'S EDUCATIONAL AND INDUSTRIAL UNION
264 BOYLSTON STREET

Dr. Christopher J. Duncan, Waban, *Chairman*
Dr. Arthur F. G. Edgelow, Springfield, *Vice-chairman*
Dr. George Van S. Smith, Brookline, *Secretary*

Round-Table Discussion: Recent improvements in obstetric technic. Dr. Raymond S. Titus, Boston; Dr. Joseph W. O'Connor, Worcester; and Dr. Arthur F. G. Edgelow, Springfield.

Section of Radiology

HOTEL STATLER

Dr. Joseph H. Marks, Newton, *Chairman*
Dr. Stanley A. Wilson, Marblehead, *Secretary*

Pancreatic Achylia. Dr. Edward B. D. Neuhauser and Dr. Sidney Farber.

Section of Physiotherapy

PARLORS D AND E, HOTEL STATLER

Dr. Henry A. Tadgell, Winchester, *Chairman*
Dr. Wilmot L. Marden, Lynn, *Secretary*

Motion-Picture Program Relating to Physiotherapy.

Section of Dermatology and Syphilology

HANCOCK ROOM, HOTEL STATLER

Dr. J. Harper Blaisdell, Boston, *Chairman*
Dr. G. Marshall Crawford, Lincoln, *Secretary*

The Local Use of Sulfonamide in Dermatoses. Dr. Harold N. Cole, Cleveland: professor of dermatology and syphilology, Medical Department, Western Reserve University.

WEDNESDAY AFTERNOON, MAY 26

Fourth General Session

GEORGIAN ROOM

Dr. Charles E. Mongan, *Chairman*
Dr. Frank R. Ober, *Co-chairman*

2:00 *Prevention of Vitamin Deficiencies in Wartime.* Dr. Franz R. Stenzel: visiting physician, Newton Hospital.

2:20 *Present-Day Rapid Methods of Treating Syphilis.* Dr. Harold N. Cole, Cleveland: professor of dermatology and syphilology, Medical Department, Western Reserve University.

2:40 *Practical Importance of the Rh Factor in Obstetrics.* Dr. Paul Gustafson: assistant in obstetrics, Harvard Medical School.

3:00 *Cancer of the Larynx.* Dr. LeRoy A. Schall: professor of laryngology and professor of otology, Harvard Medical School, and chief of otolaryngology, Massachusetts Eye and Ear Infirmary.

3:20 *Epidemic Keratoconjunctivitis.* Major Murray Sanders, M.C., A.U.S., New York City: member, Neurotropic Virus Commission, Board for the Investigation of Influenza and Other Infectious Diseases in the Army.

3:40 *Endocrine and Pseudoendocrine Problems in Childhood.* Dr. Richard Wagner: assistant professor of pediatrics, Tufts College Medical School, and physician, Boston Floating Hospital.

4:00 *The Shock Treatment in the Psychoses.* Dr. Abraham Myerson: professor (emeritus) of neurology, Tufts College Medical School; clinical professor of psychiatry, Harvard Medical School, and director of research, Boston State Hospital.

4:20 *The Medical Profession's Responsibility in the Prevention of Blindness.* Dr. Hugo B. C. Riemer: consultant ophthalmologist, Massachusetts Eye and Ear Infirmary, and supervising ophthalmologist, Massachusetts Division of the Blind.

4:40 *Blood Pressure Determinations by Patients with Essential Hypertension: Evaluation of sympathectomy over a three-year to five-year period.* Dr. David Ayman: instructor in medicine, Tufts College Medical School, and associate physician, Beth Israel Hospital; and Dr. Archie D. Goldshine: assistant physician, Beth Israel Hospital.

Scientific Exhibits

BALLROOM ASSEMBLY

Booth

S-9–10 *Anesthesia.* Massachusetts members of the New England Society of Anesthesiology.

S-11 *Technical Errors in Chest Roentgenography.* Massachusetts Tuberculosis League.

S-12 *Serving and Conserving the Nation's Health.* Massachusetts Hospital Service, Incorporated (Blue Cross).

S-13 *Your Plan for Surgical Care.* Massachusetts Medical Service (Blue Shield).

S-14–15 *First Aid in Fractures.* Massachusetts Committee on Fractures and Trauma, American College of Surgeons and Boston Metropolitan Chapter, American Red Cross. Demonstration of first aid by members of the American Red Cross Motor Corps (Boston and Newton chapters).

S-16 *Industrial Dermatosis.* American Medical Association.

S-17 *Use and Abuse of Barbiturates.* American Medical Association.

S-18 *Blood for Victory.* Blood Donor Center, Boston, Metropolitan Chapter, American Red Cross.

S-19 *Modern Pharmacy.* Massachusetts State Pharmaceutical Association.

S-20 *Surgery of the Forearm and Hand.* Second Surgical Service, Carney Hospital. Exhibitors: Dr. William E. Browne, Dr. John J. Todd and associates.

S-21–22–23 *Health in Child Care Centers.* Massachusetts Department of Public Health.

S-24 *Lesions of the Lung and Mediastinum.* Lahey Clinic. Exhibitor: Dr. Ralph H. Adams.

S-25-26 *Carcinoma of the Colon and Rectum.* Lahey Clinic. Exhibitors: Dr. Frank H. Lahey and Dr. Richard B. Cattell.

S-27 *Chemistry of Vitamin K and Synthetic Hormones.* American Medical Association.

S-28 *Prevention of Blindness.* Department of Education, Commonwealth of Massachusetts. Exhibitor: Division of the Blind.

S-33 *Venereal Disease Control.* Health Department, City of Boston. Exhibitors: Dr. G. Lynde Gately, commissioner; Dr. John T. Foley, director.

Motion Picture Program

LOWER LOBBY

Tuesday, May 25

9:30–10:00 *Single Stage Lobectomy for Bronchiectasis.*
10:00–10:30 *Occupational Dermatitis.*
10:30–11:15 *Peptic Ulcer.*
11:15–11:45 *Spinal-Cord and Cauda-Equina Injury.*
11:45–12:00 *Compound Depressed Fracture of the Skull.*
2:00– 2:45 *Modern Diagnosis and Treatment of Syphilis.*
2:45– 3:30 *Anemia.*
3:30– 4:00 *Cholelithiasis with Common Duct Stone.*
4:00– 4:15 *Thrombi and Emboli.*
4:15– 4:45 *The Story of Lyovac — Normal Human Plasma.*

Wednesday, May 26

9:30–10:00 *Mechanism of the Heart Beat and Electrocardiography.*
10:00–10:15 *Post-Partum Hemorrhage.*
10:15–10:30 *Subtotal Thyroidectomy for Hyperthyroidism.*
10:30–10:45 *Subtotal Hemithyroidectomy.*
10:45–11:00 *Lumbar Sympathetic Ganglionectomy.*
11:00–11:15 *Purposeful Splinting Following Injuries of the Hand.*
11:15–11:45 *Treatment of a Normal Breech Presentation.*
2:00– 2:15 *Indirect Inguinal Hernia: Anatomical aspects.*
2:15– 2:30 *Indirect Inguinal Hernia: Clinical aspects.*
2:30– 2:45 *Indirect Inguinal Hernia: Operative technics.*
2:45– 3:00 *Complete Laceration of the Perineum.*
3:00– 3:30 *Treatment of Asphyxia Neonatorum.*
3:30– 4:00 *Treatment of Burns of the Hand.*
4:00– 4:30 *Surgical Treatment of Varicose Veins and Ulcers.*

Technical Exhibits

BALLROOM

	Booth No.
Alkalol Company	14
American Hospital Supply Corporation	40
Ayerst, McKenna & Harrison	26
The Baker Laboratories	39
Rudolph Beaver, Inc.	53
Bilhuber–Knoll Corporation	41
Ernst Bischoff Company, Inc.	24
The Borden Company	20
Brewer and Company	9 and 10
Buffington's, Inc.	46
Burroughs Wellcome & Co. (U.S.A.), Inc.	31
Camel Cigarettes	15 and 16
Carnation Company	11
Certified Milk Producers Association	S-1
Children's Incorporated	18
Ciba Pharmaceutical Products, Inc.	48
Coca-Cola Co.	S-7 and 8
Crosbie–Macdonald	13
Davies, Rose & Company, Ltd.	47
Doho Chemical Corporation	56
Effervescent Products, Inc.	S-31
J. H. Emerson Co.	S-32
C. B. Fleet Company	51
Gerber Products Company	29
J. E. Hanger, Incorporated	61
H. J. Heinz Company	S-5
Hoffmann–LaRoche, Inc.	12
Horlick's Malted Milk Company	22
Hudnut Institute for Dermatological Research	7 and 8
Jones Metabolism Equipment Company	23
"The 'Junket' Folks"	36
Kellogg Company	49
Lederle Laboratories, Inc.	45
Eli Lilly and Company	50
M. & R. Dietetic Laboratories, Inc.	25
E. F. Mahady & Company	37–38
Mead Johnson & Company	58–59
Medical and Surgical Relief Committee of America	6
Medical Protective Company	30
Mellin's Food Company	35
E. L. Patch & Company	S-2
Pet Milk Sales Corporation	28
Petrogalar Laboratories	32
Philip Morris & Co., Ltd., Inc.	52
Picker X-Ray Corporation	19
S. M. A. Corporation	34
Sandoz Chemical Works, Inc.	44
G. D. Searle & Company	43
Sharp & Dohme, Inc.	55
Smith, Kline and French Laboratories	S-4
Spencer, Inc.	S-3
E. R. Squibb and Sons	33

The Sun-Rayed Company 17
Surgeons' and Physicians' Supply Company 60
Tailby-Nason Company 27
White Laboratories 42
Winthrop Chemical Company, Inc. 54
John Wyeth & Brother, Inc. 57

HARVARD MEDICAL ALUMNI ASSOCIATION

The annual meeting and dinner of the Harvard Medical Alumni Association will be held at 7:00 p.m. Wednesday, May 26, at the Harvard Club of Boston. Reservations *must* be made in advance through the executive secretary, Mrs. K. B. Wilson, Harvard Medical School (LONgwood 2380).

MASSACHUSETTS MEDICO-LEGAL SOCIETY

A meeting of the Massachusetts Medico-Legal Society will be held on the fourth floor of the Hotel Statler at 2:00 p.m., Tuesday, May 25 (the room number can be obtained at the Information Desk, Hotel Statler).

NEW ENGLAND SOCIETY OF ANESTHESIOLOGY

The New England Society of Anesthesiology will hold a luncheon meeting on the fourth floor of the Hotel Statler, from 12 noon to 2:00 p. m., on Wednesday, May 26 (the room number can be obtained at the Information Desk, Hotel Statler). The guest speaker will be Major Stevens J. Martin, M.C., A.U.S., of Fort Dix, New Jersey.

WAR ACTIVITIES

CIVILIAN DEFENSE

Pennant to Identify Vehicles in Blackout

A uniform system of identification of emergency vehicles to enable them to operate during real or practice air-raid alarms was announced by the Office of Civilian Defense, Washington, D. C., in Operations Letter No. 111.

The primary identifying device is a white pennant measuring 18 inches along each side with a 6-inch basic Civilian Defense insigne, that is, the letters "CD" in red inside a white triangle superimposed on a red circle. The pennant is to be attached to the left front portion of the vehicle.

To identify emergency motor vehicles at night, the operations letter further prescribes a mask to be used over the right headlight. This mask may be made of any opaque material that can be easily, quickly and securely fastened to the headlamp. It is intended for use where blackout regulations permit the use of headlights; in coastal dimout areas it should be used in conjunction with dimout equipment. The design of the mask embodies the "CD" insigne 2½ to 3 inches in diameter in green.

Vehicles entitled to use the emergency identification include the following: vehicles of the armed forces of the United States or of her allies or other vehicles acting under orders or traveling with permission thereof; vehicles of fire departments and governmental police agencies; ambulances and rescue cars and other vehicles converted to such use in emergency services; public-utility repair vehicles operating in emergency service; and vehicles in emergency service as defined by state Civilian Defense authorities.

Use of the pennants and masks described was made mandatory for the 16 states and the District of Columbia in the Eastern Defense Command in an administrative order issued by the director of Civilian Defense in accordance with the new air-raid protection regulations that went into effect on February 17. The letter recommends that all states adopt the definition of emergency motor vehicles and the methods of identification prescribed. Although many states have already adopted different methods of identifying emergency motor vehicles, it was urged that all states adopt the new devices. It was pointed out that a uniform system is particularly important in order that emergency motor vehicles that may be crossing state lines may not face unnecessary interference.

MISCELLANY

DR. ROBERTS DECORATED

Lieutenant Charles P. Roberts (MC), U.S.N.R., a Boston physician and an instructor (on leave of absence) at Tufts College Medical School, has recently been awarded the Silver Star Medal for gallantry and intrepidity in action during naval landing operations in French Morocco last November. The citation reads, in part, as follows: "The courage, leadership and professional skill of Lieutenant Roberts were outstanding and in keeping with the highest traditions of the naval service."

NOTE

Dr. William E. Ladd, William E. Ladd Professor of Child Surgery, Harvard Medical School, and head of the Surgical Service, Children's Hospital, recently gave the first Béla Schick Lecture at Mount Sinai Hospital, New York City, the subject being "Time and Choice of Operation in Early Life." This lectureship, founded in honor of Dr. Schick, formerly physician-in-chief of the Mount Sinai Hospital, has been made possible by a fund established in 1942 by his friends and associates.

CORRESPONDENCE

TUMOR DIAGNOSIS SERVICE

To the Editor: The State Tumor Diagnosis Service, which has been located since its foundation in the Huntington Hospital of the Harvard Cancer Commission, will move on May 10 to Room 387, Building E-2, Harvard Medical School. The telephone number will remain the same (LONgwood 4343). Specimen containers may be obtained from the Department of Public Health, 527 State House, as in the past.

The Tumor Diagnosis Service will continue to function as a center to aid the physicians and hospitals in Massachusetts in the pathologic diagnosis of tumors or of material suspected of origin from tumors. This service is rendered without cost. The expense is defrayed by the Department of Public Health of the Commonwealth of Massachusetts and by the Harvard Cancer Commission. Under ordinary conditions specimens will be processed and the report mailed out within twenty-four hours after being received.

Shields Warren, M.D.
695 Huntington Avenue
Boston

REPORT OF MEETING

SUFFOLK DISTRICT MEDICAL SOCIETY

A regular meeting of the Suffolk District Medical Society was held at the Boston Medical Library on October 28, 1942, with Dr. James P. O'Hare presiding. The program was a symposium on war gases.

The first speaker, Dr. Granville A. Bennett, had as his subject "Pathologic Changes Induced by Chemical Agents of Warfare." The various agents can be grouped, on the basis of their principal physiologic effects, into three main categories: vesicants, including mustard, lewisite and ethyl dichlorarsine; lung irritants, exemplified by phosgene and chlorine; and harassing agents, including the lacrimators, sternutators and screening smokes.

All lung irritants are to be regarded as acid gases. They primarily affect the respiratory tract. Chlorine produces its most pronounced effect on the mucous membrane of the upper respiratory passages, whereas phosgene injures the small bronchioles, alveolar ducts and alveoli. The principal pathologic changes are mucosal congestion with or without hemorrhage, pulmonary edema, atelectasis and emphysema. Physiologically, there are decreased aeration and impaired circulation. The injured lungs are susceptible to secondary infections, notably bronchitis and bronchopneumonia.

The vesicants injure any tissues with which they come in contact either in liquid or in vapor form. Mustard gas causes little or no immediate irritation. It produces skin burns that lead to progressive vesicle formation over a period of twelve to thirty-six hours; these vesicles usually reach their maximal size in forty-eight hours. The vesicular fluid is clear and contains no trace of the agent. The burn causes much less discomfort than a thermal burn of similar extent. The lesion does not extend below the subcutaneous fat. Healing is slow. Lewisite acts much more rapidly than mustard gas and causes more marked symptoms of pain and tingling. Furthermore, the lesions penetrate to a great depth. If applied to the skin of the abdomen of a rabbit, the agent produces burns that extend into the peritoneal cavity and affect the liver or intestines. The vesicants produce pronounced necrotizing lesions in the upper respiratory tract. Secondary infections in the lungs, especially multiple lung abscesses, are prone to develop.

The treatment of persons contaminated with vesicants must be carried out immediately. Unless the agent is removed or neutralized within five minutes, a burn will develop. After fifteen to thirty minutes, prophylaxis is ineffective. The liquid agent should be quickly sponged off with any absorbent material at hand. Care should be exercised to prevent spreading the agent. A neutralizing agent should then be applied. Any commercial laundry bleach (sodium hypochlorite) may be used for mustard gas. Hydrogen peroxide, preferably 8 per cent strength, should be used for lewisite. These agents should not be used in the eyes, which should be washed quickly with running water or irrigated with a 1.5 to 2.0 per cent solution of bicarbonate of soda.

Dr. Bennett described the construction and operation of cleansing stations such as may be needed at hospitals to receive injured and contaminated casualties.

Dr. Andrew Contratto discussed the topic "Diagnosis and Early Treatment of Pulmonary Gas Casualties." The primary effect of gases is on the pulmonary system, the cardiovascular manifestations being secondary. Phosgene is the most likely gas to be used. Its odor, which is the best means of identification, is like that of musty hay. Early, there may be slight eye irritation, cough and nasopharyngeal irritation. In heavy doses there may be nausea and vomiting. Often the victim may be aware of nothing, unfortunately. There is a latent period of four to forty-eight hours. Cough is usually present after about nine hours and is soon followed by cyanosis. The appearance is that of cardiac failure, with frothy sputum, pulmonary edema and rales, and asthma. The treatment consists of immediate and absolute rest in bed as soon as it has been established that a person has been exposed. Physiologically, there is early hemoconcentration. The degree of this phenomenon is a good prognostic index. The plasma protein is unchanged or diluted, which probably indicates extravasation into the areas of the pulmonary edema. The latter finding accounts for most of the manifestations. The stage of blue cyanosis is the equivalent of hemoconcentration. Therefore, treatment should consist of inhalations of 100 per cent oxygen, venesection of 500 cc. (several times a day, if necessary), bedrest, heaters, the Trendelenburg position, the use of digitalis if the patient is old or known to have cardiac disease, and the use of aminophyllin if he is known to be asthmatic. The use of morphine is still a moot point, but the drug can probably be employed in small doses. The onset of gray cyanosis, which may even be primary if the hemoconcentration is sufficiently high, is akin to clinical shock and is invariably a premonitory sign of impending death. Treatment is similar to that for shock. In phosgene poisoning 80 per cent of the deaths occur in the first twenty-four to thirty-six hours, whereas in mustard-gas poisoning only 3 per cent of the deaths occur in a similar period.

Dr. Maxwell Finland next discussed the subject "Secondary Pulmonary Infections." Previous reports show that 22 per cent of the deaths from mustard gas occur on the fifth day, whereas 60 per cent occur in the next five days. Infection plays a definite role in at least 75 per cent of these cases. Heart failure is often a factor and may be precipitated by pulmonary fibrosis. Mustard gas frequently leads to chronic bronchitis and emphysema, with disability for eight to ten years. The question whether tuberculosis may be a result is still unsettled, but there is some evidence that arrest or quiescent cases may be activated. Delayed pulmonary irritation is especially likely to lead to secondary infection. Acute and persistent pulmonary disease is particularly prevalent in those who have been gassed. Fibrosis appears to be a great factor. The changes seen in such lungs are similar to those found following the pandemic of influenza. The most commonly encountered bacteria are the hemolytic and indifferent strains of streptococcus, and the higher types of pneumococcus, which comprise the usual mouth flora. Staphylococcus is relatively rare, but when present leads to the serious sequelae of necrosis, abscess and bronchiectasis. Much depends on the past history of the patient regarding exposures. The secondary invaders may follow an epidemic, such as one of measles, streptococcus throat or pneumonia. The treatment is the same as for any secondary infection. Early recognition depends on an elevation of the temperature, of the white-cell count and of respirations and on increased malaise. Chemotherapy should be given early, since the mortality may be lowered by diminishing the number of secondary infections. This type of treatment may also decrease the number of chronic cases, the complications and the spread. The drugs of choice in staphylococcal infections are sulfadiazine

and sulfathiazole; in streptococcal infections, sulfapyridine should be used if sulfadiazine is not available or has failed. To avoid secondary infections, gas cases should be segregated in wards, and the patients should also be protected against infection carried by the ward personnel.

Dr. David G. Cogan spoke on the topic "The Ocular Effects of War Gases." The irritant gases are potentially bad for the eyes but not in the concentrations met in the field. The only serious gases are mustard and lewisite. Man is somewhat protected by tears. The reaction to mustard vapor, which was frequently used in World War I, is characterized by a latent period of four to six hours. The liquid may be sprayed from a plane and cause a more serious "splash" burn. The result of a vapor burn is a severe conjunctivitis, with swelling out of proportion to the redness. This begins to subside in eighteen to twenty-four hours, and there are no sequelae if the vapor concentration was not too high. A splash leads to an early, atypical corneal opacity, with recurrent exacerbations for months or even years. Such patients almost always apply late for treatment because of the latent period. Early therapy, which is useless unless applied within fifteen to thirty minutes, consists of irrigations with olive oil. But in the absence of this material, one should use any fluid rather than wait. Lewisite causes almost immediate manifestations and so forces the person to earlier treatment. But even this is often ineffective, because the effects of the agent are almost instantaneous.

BOOK REVIEWS

The Pathology of Trauma. By Alan R. Moritz, M.D. 8°, cloth, 386 pp., with 117 illustrations. Philadelphia: Lea and Febiger, 1942. $6.00.

Traumatism is of growing importance as a major hazard of living. The machine age with its advances in methods and speed of transportation, and its mass production, has added to the jeopardy of ordinary life by increasing the chances of exposure to mechanisms that cripple or kill impersonally. War is waged by violence applied with malice aforethought, as illustrated in the commando's hundred ways to kill. From any viewpoint the study of the effects of violence on the animal body has become a matter of primary import. Though certain effects of traumatism, for example, fractures, have been given adequate attention, there is no branch of medicine in general that has been more neglected. Traumatic problems have been referred casually to the school of forensic medicine, as things apart from general medicine. Because forensic medicine has tended to be static in some relations, much of our "knowledge" of the effects of traumatism is of legendary inheritance.

Dr. Moritz has not only helped to bridge a hiatus in medical literature but has also replaced the confused explanations of some phenomena by scientific information that clears the atmosphere. The book is well planned. It deals, first, with types of traumatism and the effects of trauma on tissues in general. The relation of traumatism to possible causation and aggravation of tumors is well covered, as is the relation of trauma and infection, immediate or delayed. These general chapters are followed by discussion of the effects of violence on special organs or organ systems, that is, the circulatory, respiratory and nervous systems, the abdominal organs and the genitalia, together with a chapter on injuries to bones, joints, bursas and muscles.

The work is well illustrated and well documented with a modern bibliography following each of the ten chapters. It can be recommended without reservation not only to surgeons, to students of forensic medicine and to pathologists, but also to clinicians who are interested in the basic problems underlying the practice of medicine.

Blood Grouping Technic. By Fritz Schiff, M.D., and William C. Boyd, Ph.D. With a foreword by Karl Landsteiner, M.D., Sc.D. (hon.). 8°, cloth, 248 pp., with 45 illustrations and 67 tables. New York: Interscience Publishers, Incorporated, 1942. $5.00.

This small book is a systematically arranged manual dealing with the technics of blood grouping and their special applications to transfusions and to medicolegal and anthropological problems. Being completely devoid of padding, it is well suited for teaching and laboratory reference. Originally a modification in English of the late Dr. Schiff's well-known German work, it has had considerable revision at the hands of the American author, Dr. Boyd. A small but up-to-date section on transfusions, blood banks and blood substitutes is provided. Complete directions are given for the use of the M and N factors in bastardy cases, and the newer knowledge regarding the Rh factor, "fractions" of the A and B agglutinogens and so forth are presented. There is a rather amusing section concerning what constitutes an "expert" in blood grouping.

Although the book is not nearly so comprehensive as that of Alexander Wiener, it is often more specific in its technical directions, and is therefore a necessity in any laboratory that attempts to do more than simple blood grouping and cross-matching.

Dr. Finley Sees It Through. By Alan Hart, M.D. 8°, cloth, 370 pp. New York: Harper and Brothers, 1942. $2.50.

This is an interesting story of a physician living in a small town and taking an active part in all the medical affairs of his environment. With it is interwoven the story of a romance and its effect on the doctor's career. There is much of interest in this simple story. The medical aspects are well covered by the author, who graduated from a well-known medical school in 1917 and practiced first in Montana and later in New Mexico. He writes with unusual fluency, and his book is better than the average medicated novel. On the other hand, it is too medicated to be literature of importance.

Medical Leaves: A review of the Jewish medical world and medical history. Volume IV. Edited by Dr. Hershel Meyer. 4°, cloth, 223 pp., with 12 illustrations, 21 tables, 3 graphs, 2 figures and 7 portraits. Chicago: Medical Leaves, Incorporated, 1942. $3.00.

This collection of twenty-five articles has much interest to medical historians. There are short biographies of eminent Jewish physicians, including Delmedigo, Hafkine and DeCyon. Dr. Savitz writes about Dr. Hyman Morrison, of Boston, on the occasion of his sixtieth birthday. There are sketches of the Mt. Sinai Hospital in Baltimore, of the Montefiore Hospital in New York City and of the Hadassah University Medical Center in Palestine. There is an important contribution by Professor Hirsch on the causes of death among the Jews. Dr. Ulrich Friedemann discusses the problem of the Jewish physician immigrant, and there are several articles on the medical education of the Jews.

(Notices on page xii)